This magazine is published to promote sound thought upon and concerning industrial medicine and traumatic surgery. To that end it will contain articles, news items, reports, digests and other presentations, together with editors' comments. The editorial policy is to encourage frank discussion. On this basis contributions are invited.

Industrial Medicine

Reg. U. S. Pat. Off.

The editors will exercise care in checking on the accuracy of data printed, but in all other respects articles and opinions of which expression is allowed are the opinions of their authors—the editors reserving in all cases the right to comment on the same, in the current or any subsequent issues, as they may be inclined.

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The Neurological Component

—*of a General Examination*—

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Chicago

A NEUROLOGICAL examination is necessary at the time of employment, in periodic examination, in intercurrent disease and injury, for purposes of decisions concerning compensation, and insurance and retirement or pension.* At each of these several times the conscious or unconscious wish of the person examined differs and gives rise to difficulties in diagnosis. Whereas during the draft of the World War, soldiers requesting examination for reasons of purported disability entitling them to discharge, had a desire to avoid military service, applicants for positions hope for employment. In the first instance it was necessary to detect malingering, in the second concealment of disease and disability. When first suffering from disease or injury the patient is anxious to assist the physician and neither hypothecates symptoms nor conceals them; later for purposes of compensation conscious or unconscious wishes induce the patient to do both; whereas in periodic examinations and for retirement for disability there is a tendency to conceal or minimize a disability.

Since at the time of employment it is the wish of the person to appear to be in good health, the indications for the examination are to discover concealed or unrecognized disease and disability. Were it always possible, by the elicitation of objective signs, to discover disease or disability, the exclusion from industry of those who are unfit would be relatively simple. Unfortunately this is not the case. A number of organic diseases of the nervous system may be recognized by objective signs. Such diseases as the residuals of anterior poliomyelitis, of cerebral palsies, of multiple neuritis, the muscular atrophies, tabes dorsalis, dementia paralytica, multiple sclerosis, lesions of the spinal cord, among many others, may be cited as examples. Certain other conditions, as epilepsy, are not so easily discovered. Particularly difficult is it to determine at such an examination, the existence of a mental deficiency, a psychopathic personality, a neurosis or psychosis. It is unfortunate that it is these very conditions that militate against successful industrial life. The stupidity of the mental defective makes it necessary to expend much time unsuccessfully in attempts to teach him his duties which he will never adequately perform; he will always be a source of potential danger in relation to injury to himself or others by reason of his faulty judgment, carelessness, and often slow reaction time and other motor defect. Far more costly are the psychopaths, oftentimes superficially bright, who with emotional instability and inadequate personalities, are unreliable, querulous, litigatory, quarrelsome, suspicious, truculent and insubordinate, often given to sabotage, incapable of sustained effort, frequently changing places of employment, consistently indulging in absenteeism, at times civil offenders. The psychoneurotics are incapable of adjusting to difficulties, taking refuge in functional disorders, unable to perform arduous and sustained labor, disposed to the development of traumatic neurosis and valetudinarianism following minor injuries. It is unnecessary to point out the dangers of employing a psychotic applicant.

Some indication of the importance of neuropsychiatric conditions in the rejection of applicants may be obtained from the statistics of the Surgeon-General of the various types of nervous and mental diseases which disqualified for military service during the Great War, leading to discharge. As of May 1, 1919, there were 69,394 such cases. Thirty-one per cent of these were mentally deficient, 17% suffered from neurosis, 11% were psychotic, 10% had organic diseases of the nervous system, 9% were psychopaths, 9% were epileptics, and 6% were addicted to drugs including alcohol; 7% suffered from some glandular disorder and should properly be classified under visceral disease. The number of soldiers examined is difficult to estimate, so that the percentage of these individual groups in the Army cannot readily be determined. The largest number of the cases leading to military disqualification then are the feeble-

* Presented at the Forty-Ninth Annual Congress of Railway Surgeons, Chicago, September 19-21, 1938.

minded, then the neurotics, then shortly following the organic diseases of the nervous system, the epileptics and psychopaths. Ten per cent (the organic diseases) suffered from conditions recognizable by the elicitation of objective signs and 83% from conditions no so recognizable.

THE widespread existence of mental deficiency is emphasized by the statistics of the combined draft and camp examinations where it was found that there were registered 10,101,506 men between the ages of 21 and 31 years; in 12.06 out of every 1000 men examined the individual was unfit as a soldier because of mental deficiency, giving a total of 121,824 men rejected. Inasmuch as the standard of rejection in the Army was low, such figures must be multiplied considerably if we would include the dull, stupid borderline cases probably equally unfit for successful industrial life unless properly classified and directed. It would appear that a psychometric examination of applicants would be a profitable procedure. How this would best be effected, for example by group tests, is beyond the scope of this paper. Indications for a psychometric examination in individual cases are more often found after than before employment when carelessness, inability to learn and understand orders and such characteristics as gain for such a person the name of boob among his fellow workmen, become evident. However, at the time of the first medical examination, the general conversation necessary to conduct a physical examination often will reveal defects in learning, reasoning, language and judgment. Untidiness, lack of personal cleanliness, and muscular awkwardness may call attention to a possible mental defect and at times anatomical stigmata of degeneration may be found. Of great importance is the history of school and vocational career. Of all the neuropsychiatric groups the mental defectives show the least progress in education. Of the cases studied by psychologists in the draft boards one-third of the whites and two-thirds of the colored had no education at all. Despite the fact that they were dealing with higher grades of defectives, only a small number reached high school and 59% had some schooling. Since the group is not amenable to learning, their industrial history gives evidence of short periods of employment, failure to advance and many periods of unemployment and frequent change of positions.

The psychoneuroses frequently escape detection at routine examinations. Desiring occupation, pertinent facts of preceding illness are withheld and the condition is discovered only some time after employment when, under stress of circumstances, the symptoms come to the front. When a history is obtained of periods of illness characterized by symptoms which should be related to some visceral or other disease residuals of which should be demonstrable and which are absent, a presumption may be made of the existence of a psychoneurosis, and more intensive search instituted. The ordinary complaints of anxiety, compulsions, marked doubts, fears, and hypochondriasis will not be voiced, nor will the stigmata of hysteria be found. However, shyness, timidity, tremor, tachycardia, hyperidrosis, vasomotor disturbances of flushing, coldness and cyanosis of extremities, dermographia, stammering, increase of startle, and polyuria may give some indications of the existence of such states. Since it is characteristic of the group that they break down soon after meeting difficult situations, the industrial history reveals employment periods of short duration. If obtainable, the history reveals a family of neurotic taint.

In the 4,500,000 men examined during the war, approximately 15,000 cases of insanity were discharged or rejected, a ratio approximately of three per 1000. A smaller percentage of psychoses were discovered at the routine neuropsychiatric examination than of any other clinical group. This is partly due to the fact that continued observation is so often necessary for the recognition of mental disease. In industry those who are deeply depressed and suicidal or similar to those who in military service came to the attention of physicians because of delusions of persecution, strange attitudes and actions, are unlikely to seek employment. Others may, in whom indifference, apathy, inappropriate emotional reactions and lack of connectedness in conversation, or emotional outbursts may call attention to a possible dementia precox or mild depression. Lack of interest, retardation or talkativeness, exhiliration and overactivity call attention to a possible manic-depressive psychosis. Since, unless a frank history of the symptoms is available, an observation period is usually essential for discovering mental cases, many undoubtedly enter industrial life only to be discovered later.

Practically the same proportion of constitutional psychopaths as of the psychoses were recommended for discharge. This group is composed of men who are unstable, undependable, ill-balanced emotionally, changeable, and in general asocial. They are unable to carry out the requirements of organization. A considerable number in the Army had criminal records and a tendency to recidivism, to do the same thing over and over, whether the act was antisocial or not and whether repeatedly punished for it, was pronounced. They were for the most part fairly normal in intellectual performance and presented few physical signs. They were recognized chiefly by their past records. In this respect it is obvious that complete records, such as are obtained through social service assistance, are invaluable for the exclusion of such undesirable individuals from industry. The classification of these psychopathies into groups of criminalism, nomadism, inadequate personality, etc., is possible only in a very intense inquiry and probably is not possible at the time of a single examination. Since the condition is constitutional in nature if a history is obtainable, both the school and industrial record give evidence of asocial behavior.

Epilepsy constituted about 9.2% of neuropsychiatric conditions leading to discharge. With the exception of the psychoses, the epileptic group had the smallest percentage found in routine examination by neuropsychiatrists. This means that with-

out a history of previous attacks it is often impossible to detect the existence of an epilepsy; therefore, they are discovered only after an attack has been observed after induction into military service or industry. A small percentage of the epileptics are due to residuals of disease of the brain or to some active disease. Among these may be mentioned injury at birth, toxic or infectious encephalitis, encephalopathies, epidemic cerebrospinal meningitis, cerebral hemorrhage, thrombosis, embolism, tumors, diseases, etc. For the most part in such cases objective signs can be discovered of weakness, paresis or paralysis, optic atrophy, extra-ocular muscle palsies, spasticity, contractures, ataxia and other dysfunctions which will be discussed with organic diseases of the nervous system.

A very small number suffer from tuberculous sclerosis of the brain and may show tuberculous and acneform eruptions of the skin, particularly the face, associated with mental dullness.

In those suffering from idiopathic or functional epilepsy it is often impossible to find evidence of the existence of the condition. Scars of injuries sustained in previous seizures may be discovered about the head or face or other parts. Occasionally scars of burns obtained during seizures and scars in the tongue from tongue bites may be seen. I have been unable by voice or conduct or appearance of most patients suffering from epilepsy to so discover them.

OF THE cases discovered after induction into the Army, organic disease of the nervous system exclusive of the epilepsies and including general paresis, constituted 10% of those discharged for neuropsychiatric disease. Of these, diseases due to syphilis, paresis, cerebrospinal syphilis or tabes constituted 4.8% of the total, almost one-half of the organic diseases of the nervous system. Cerebrospinal syphilis constituted the largest group, then multiple sclerosis, paralysis (unclassified), injury to the brain, tabes dorsalis, chorea, hemiplegia, tremors, tics, neuritis, poliomyelitis, sciatica, then smaller numbers of nervous diseases, progressive muscular atrophy, paralysis agitans, etc. The classification used in the Army was poor but the most important conditions were syphilis of the nervous system, residuals of disease or injury to the brain, usually producing hemiparesis of paralysis, multiple sclerosis, poliomyelitis, acute and chronic, etc. It is impossible in this short time, and probably unnecessary, to describe the technic of a neurological examination. A minimum of such an examination should, however, constitute a part of the general examination. It should include an examination of the cranial nerves, including rough visual fields, vision, ophthalmoscopic examination, function of extra-ocular muscles, pupillary reactions, and hearing, and examination of motion for weakness, paresis, paralysis, and for increased motion as spasms, tremors, tics, dystonias, and spasticities, of sensation, of deep, superficial, pathological and sphincter reflexes, state of muscles, flaccidity, atrophies, hypertrophies, coordination and speech.

It may be of interest to note some of the easily observed defects calling attention to some of the diseases noted above.

Inspection of the pupils often calls attention to the existence of neurological disease. Inequality, irregularity, fixation and the Argyll-Robertson pupil all call attention to such conditions. One must keep in mind that unequal pupils need not mean syphilis or other disease of the central nervous system but may result from a lesion of the cervical sympathetic chain of one side, due at times to a non-disabling condition, such as scars from inflammatory diseases, apical pleurisy, etc. Irregular pupils may be the residue of an iriditis as well as a fixed pupil. Argyll-Robertson pupils, however, usually indicate tabes dorsalis, paresis, at times polio-encephalitis, infectious or alcoholic.

A rough visual field is easily and quickly obtained. No other disability may be demonstrable than a homonymous hemianopsia, in a tumor or other lesion of the occipital lobe and a homonymous hemianopsia may be the first sign of a pituitary adenoma. An ophthalmoscopic examination is essential in a general examination. Primary optic atrophy may call attention to a tabes or paresis; a pallor of the temporal halves of the discs to a multiple sclerosis, a papilledema to increased intracranial tension, as from tumor.

Although strabimsus of very early onset may not be related to disabling neurologic disease, complete paralysis of the muscles supplied by the third, fourth and sixth nerves is so indicative, and the late onset of a weakness of an extra-ocular muscle quite significant. Nystagmus calls attention to lesions about the cerebellar peduncles or brain stem, and is found often in multiple sclerosis, cerebellopontine angle tumors, acoustic neuromata, or cerebellar disease. Upward nystagmus is in my experience always due to a lesion in the brain stem. Of course, muscle imbalance produces slight nystagmoid movements on extreme lateral gaze and must be properly evaluated. Failure of convergence may indicate neurological disease but one must remember that a disproportionately far "near spot" may result in failure of convergence of that eye, and be the expression of an amblyopia ex anopsia and not due to a neurological disease. Unilateral nerve deafness calls attention to a possilbe acoustic neuroma, or a bilateral one to syphilis.

Neurological diseases leading to increase of motion are easily detected upon inspection. Tics, such as blepharospasm, torticollis, etc., are apparent to cursory inspection. Choreiform movements are also easily seen but sometimes it is necessary to have the patient stand on one foot with extended arms and the tongue protruded when movements may be discerned in the upper extremities and always in the tongue. Tremors are seen in such diseases of the basal ganglia as paralysis agitans or sequelae of encephalitis. They occur with the muscles at rest, often cease temporarily when the extremity is moved passively or actively, usually are slow and rhythmical. In multiple sclerosis or other diseases producing lesions in the brain stem or cerebellum peduncles intentional tremor occurs.

At rest no tremor may be observed but upon intentional movement, as touching the nose with a finger, it may occur during the movement or upon attempts to keep the finger approximated to the nose. Pouring water from one test tube into another is an excellent way to elicit an intentional tremor. Generalized tremors, including the tongue and lips, occur in paresis.

Weakness may ordinarily be determined by the patient's resistance to passive extension of a voluntarily contracted muscle. The residuals of a hemiparesis or a beginning one may be brought out by the following observations. At times the palpebral fissure on the affected side is larger, the nasolabial fold more shallow, and on attempt to forcibly show the teeth the platysma on the affected side does not contract. When the arms are outstretched and the eyes are closed, when the patient elevates and lowers one extremity the other being held firmly outstretched, as the normal one is elevated, the affected one sinks so that after a number of repeated movements the affected arm may have dropped almost completely. In the lower extremity, when the patient attempts to get up from a recumbent position without the assistance of his arms, the affected leg often raises from the examining table. An inspection of station and gait often calls attention to neurological disease. Standing or hopping on a defective extremity may be clumsy or impossible. In chorea, however, the patient can stand better upon the affected side. An increase in ataxia on standing with the feet together when the eyes are closed calls attention to a sensory ataxia found in tabes. Of course, the ordinary hemiplegic and both spastic paraplegic and steppage of flaccid paraplegic gaits are easily detected. Very early in the development of Parkinsonism the normal associated swing of the upper extremities in walking disappears and on the affected side it will be seen that the patient does not swing his arm but holds it adducted and flexed at the forearm.

Increase or diminution of all reflexes may have no significance. But a disproportionate increase of deep reflexes in the lower extremities might. The increase of deep reflexes on only one side and a diminution of superficial reflexes on the same side are particularly significant. Absence of the deep reflexes, Achilles and knee jerks may indicate a tabes dorsalis. A loss of deep sensibility or pupillary changes may confirm the diagnosis. Loss of the deep reflexes alone is not indicative of neurological disease. Absence of the abdominal reflexes, although normally seen, is an early sign of multiple sclerosis. The existence of a Babinski or other pathological reflexes, of course, at once calls attention to neurological disease. The state of the muscles should always be determined. The patient should always be completely disrobed. Atrophy in groups of muscles supplied by peripheral nerves or in parts, as below the elbow and below the knees or in distribution of spinal segments calls attention to neurological disease. As a residue of poliomyelitis atrophy occurs in the distribution of spinal cord segments, as deltoid, biceps, brachioradialis. In progressive muscular atrophy it is more widespread but also spinal segmental. In the ordinary types Aran-Duchenne, the small muscles of the hand are very early affected; in the Charcot-Tooth-Marie type, the extremities below the elbow and knee. During the development of a progressive muscular atrophy fibrillary twitching of the muscles may be seen and at times an amyotrophic lateral sclerosis is ushered in by a period of widely disseminated fibrillary twitching preceding weakness and atrophy by several months. The discovery of loss of sensation is sufficient to determine the existence of neurological disease and it is needless to point out the characteristics of sensory loss in nervous diseases as the dissociation between loss of pain, temperature senses and conservation of touch in intramedullary disease, as syringomyelia, etc.

To what degree industry suffers from its inclusion in its ranks of those who are affected with neuropsychiatric disorders cannot be estimated. From the lessons learned in the Great War, it would seem it suffers to a considerable extent. It would seem important to devise a system whereby such persons were early detected by industry as a whole, excluded from competitive industry and provision made for their care or limited and directed occupation elsewhere.

Discussion:

DR. ROLAND P. MACKAY, Chicago: Dr. Pollock's comprehensive treatment of his subject leaves me only the discussion for emphasis of certain points which he has brought out already.

In the first place, while it probably is not desirable that a complete neurological examination be made upon every patient who comes to you, it is true that in any general examination of a patient there are certain indispensable neurological elements. Since a general examination is designed to detect the presence of any form of disease in a patient, one cannot stress too much what Dr. Pollock has already indicated, namely, that history taking is in itself a part of the examination. For example, a detailed inquiry into the family history, marital, past medical and surgical history gives us certain facts about the background of the patient, but his memory, his attention, the degree to which he can maintain attention, his expression and various types of dysarthria may also be ascertained. Often an emotional disturbance will appear under such inquiry. It is deporable that too often this important matter of taking the history is left to the hands of clerical assistants, nurses, etc. It is very easy for a young nurse to ask a patient whether he gets up too often at night to pass his urine and get a true answer, but it is very difficult for this nurse to detect the fine shades of emotional response when he is questioned, for instance, about his deceased wife. Therefore, it seems very important, in so far as possible with a patient in whom a neurological disturbance is suspected, that a physician take the history, because the history taking is really a part of the neuropsychiatric examination.

One point that occurs to me is that the general physician all too often follows certain stereotyped medical clichés in examinations. It is well known that the knee jerks will be lost in tabes dorsalis, and I suppose that if I have seen one I have seen 50 patients examined by various physicians in whom the knee jerks had been tested and found present but in whom there was a definite tabes dorsalis when attempts to elicit ankle jerks were made. In practically every case of tabes dorsalis the ankle jerks are lost years before the knee jerks are lost.

Again, we physicians oftentimes expect in severe cranial nerve injury that the patient will show some objective neurological abnormality, whereas that is often

not the case. Instead we see a very definite stereotyped symptom complex which comes out in questioning the patient; namely, that the patient has a postural type of vertigo; that he has a headache brought on by these changes in posture. There may also be certain personality changes which are apparent only on taking the history.

Again we very often expect to find objective findings in cerebrospinal syphilis. All too often syphilis of the nervous system is latent. Very often we have patients in whom we have no neurological abnormalities but there is a definite syphilis of the central nervous system. One of the most important things we can do is to insist that routine, repeated Wassermann examinations be made on all patients. This is especially true in railroad surgery because, as we know for reasons not necessary to go into at present, railroad employees appear to be particularly liable to have syphilis. Free use of the spinal puncture should not be left unemphasized, but we must remember, before doing this, that the patient must have a careful ophthalmoscopic examination by a competent man.

Finally, I want to emphasize that the neurological examination is constantly being refined. It is not necessary to do only those things that Charcot, Gowers or Hughlings Jackson taught. It is necessary to keep up to date on the new methods brought out every day. For example, it is not inadvisable to diagnose epilepsy in a patient with repeated attacks of unconsciousness with convulsions because we now know many of these patients have a hyperirritable sinus reflex complex, and this can be determined by a very simple procedure. Orthostatic hypertension may also give rise to unconsciousness.

In recent years we have become familiar with narcolepsy. Unless you are familiar with this condition you are not equipped to evaluate the history and findings in these patients.

Now another field that is important in neurology, and which is going to be more important as the years go by, is the vegetative nervous system. The surface has only been scratched in this part of our science.

The important thing about all this is to be suspicious; never be easily convinced. If you suspect the presence of a neurologic or psychiatric abnormality, you can at least call in the aid of a neurologist or someone who is a specialist in that field to confirm or deny your suspicions. In examining these patients, particularly in the psychiatric patients, it is necessary to be very ingenious. I shall tell a story about Dr. Pollock which indicates how ingenious a man must be to get an estimate of a person's intelligence. During the World War, Dr. Pollock was called upon to examine a great many soldiers. I believe he was down in the Kentucky mountains or examining men from that region and had to examine many mountaineers who had had very little formal training and were not susceptible to examination by the Binet-Simon test, so Dr. Pollock had to do the best he could. Up comes a lanky, tall mountaineer and Dr. Pollock asked several questions about where he lived, about his family and how far it was to certain places, and finally he said, "Can you tell me the difference between a bull and an ox?" and the man said: "Yes. God made the bull and dad made the ox." It is not necessary to give a long test to such a patient as that.

I wish to express my appreciation to Dr. Pollock for his comprehensive discussion of this difficult subject and my appreciation in having been asked to discuss the paper.

DR. HARVEY BARTLE, Philadelphia: I recall some few years ago we had a large turn-over of employees and we wanted to have a personality study or a psychological study of our people. With my neuropsychiatrist we developed a seven-point program, one that did not require a lot of time, but you would be surprised growing out of that the percentage of rejections purely on that examination. When we developed the scheme for approaching the subject, I went to a celebrated industrial man and asked him what he thought of it. He suggested that we turn it over to a psychologist and then he suggested that we try it for six months and see how it worked. It is working fine. I think it is our responsibility to recognize these early conditions. We must recognize these trends and refer the patient to a neurologist.

Central States Society

—Program of the Annual Mid-Winter Clinical Meeting, Milwaukee, Wisconsin, December 9, 1938—

THE Central States Society of Industrial Medicine and Surgery (component society of the American Association of Industrial Physicians and Surgeons) will hold its Annual Mid-Winter Clinical Meeting at the Pfister Hotel, Milwaukee, Wisconsin, during the day and evening of Friday, December 9, 1938.

The Program Committee under the chairmanship of DR. URBAN E. GEBHARD, of Milwaukee, has the following members: DR. WALTER J. JONES, La Crosse; DR. L. E. FAZEN, Racine; DR. JAMES A. JACKSON, Madison; DR. RALPH M. CARTER, Green Bay. They have prepared the following program:

MORNING:

9:30 "Surgical Problems Involving the Os Calcis"—DR. EDWARD QUICK, Milwaukee.

10:00 "Traumatic Abdomen"—DR. S. H. WETZLER, Milwaukee.

10:30 "Infections of the Hand"—DR. J. B. MATTHEWS, Milwaukee.

11:00 "Industrial Examinations"—DR. A. W. GRAY, Milwaukee.

11:30 "Silicosis and Silico-Tuberculosis"—DR. O. A. SANDER, Milwaukee.

LUNCHEON

AFTERNOON:

2:00 Address—DR. WILLIAM C. GOENNE, President, Davenport, Iowa.

2:30 "Estimating Disabilities"—DR. RALPH M. CARTER, Green Bay.
Discussion by DR. A. J. WEBER, Milwaukee.

3:00 "Common Errors in Treating Fractures"—DR. KELLOGG SPEED, Chicago.

4:00 "Back Injuries and Their Treatment"—DR. MERRIT JONES, Wausau.

5:00 "Discussion Injection Treatment of Hernia"—DR. FREDERICK W. SLOBE, Chicago.

EVENING BANQUET:

6:30 DR. WILLIAM C. GOENNE, President, presiding.
Guest Speaker: DR. HARRY E. MOCK, Chicago—"Diagnosis and Indications for Surgery in Head Injuries."
General Discussion.

All industrial physicians and surgeons and all members of the medical profession interested in promoting advances in traumatic and industrial surgery are cordially invited to all of the sessions. There is no registration fee.

The Wisconsin members are to meet separately at luncheon to complete plans for organizing a State Society.

The Hernia Situation

—At a Plant Employing 1000—

CARLOS E. FALLON, M.D.,
Newburgh, New York

THE greatest problem confronting us at the plant where I am employed is the so-called traumatic hernia case. Some of these cases are clear cut compensation cases, others are questionable. However in the New York State Compensation Court the employee is invariably right, and we have found it best to care for most of the questionable cases under some plan for operation.

The old problem of whether hernia is a congenital weakness or a true new traumatic hernia has given a great deal of food for thought. From our records we believe there is a congenital weakness or no hernia will develop.

Having examined our group of 612 to 1419 employees once a year for 10 years, I have decided that it is not best to use any pressure in examining rings, and it is very rare that a large ring is not detected each year if present. A method of recording inguinal rings from year to year can be uniform by listing as "0" a ring which will not admit the tip of the little finger; a Size 1 ring as one which will admit the tip of the little finger, and a Size 2 ring as one which admits the tip of the little finger and tip of the index finger. The Size 2 inguinal ring is enlarged so that there is an impulse on coughing but no protrusion of the abdominal viscera. Another point I believe should be considered is that the size of inguinal rings can vary slightly from time to time, depending on the type of work being done, and the general health of the individual at the time of examination. I am confident I have detected a large ring on a man doing heavy lifting work, and having sickness in his family necessitating his losing his rest, which appeared normal six months later when the employee had been transferred to a clerical job and had had plenty of rest.

Our plant is thoroughly equipped with monorails and every device to eliminate liftings. It is my experience, however, that many hernias develop when the employee is hurrying and tries to make time by not using the equipment he has furnished for him.

There are 48 employees in our plant with definite large inguinal rings. These men all have been advised that they have large rings, and that if they are not careful, they will develop hernias. We have also tried to place them in jobs where there is as little lifting as possible. There have been 32 hernias repaired during my 10 years of service, with five recurrences. Three recurrent hernias have been repaired by fascial repair method and the men changed to some other line of work. Two men refused second repair and are wearing company-furnished trusses. Including these two there are 16 hernias not operated, among our employees. Most of these are old men in non-lifting jobs or office employees who prefer to wear a truss rather than have an operation.

We have had several very peculiar hernia situations, a few cases of which I shall describe.

CASE No. 1 I relate because there was no external trauma at the site of injury. MR. P. C., age 48, was struck on the back of the head by a sliding ladder and knocked unconscious; upon recovering consciousness he had an indirect inguinal hernia. Evidently the shock of being struck caused a dilatation of the lower abdominal organs, resulting in increased intra-abdominal pressure, which broke the hernia through. This was repaired and, since the employee asked to be returned to his work as a fireman in the boiler room, he was allowed to return there. Two years later he had a direct inguinal hernia develop gradually on the same side, and has refused to have a corrective operation.

CASE No. 2 shows the small amount of pressure which may result in a hernia. MR. H., 64, who is now on pension, had enlarged inguinal rings on both sides. For some time he had been troubled with nocturea and his wife, who is 15 years younger, told him she thought his nocturea was purely a habit condition and that he should try to sleep through the night and not go to the bathroom so often. One night he tried not getting up and when he came to work the following morning he had an indirect inguinal hernia of the right side, the size of a small orange. He was pensioned soon after, and wears a truss rather than having a hernial repair.

CASE No. 3, MR. R. M., 41 years of age, struck his left lower abdomen on the handle of a machine January 26, 1937. February 2 he reported that he had had a great deal of intestinal distress since the accident, but upon examination there was no external evidence of injury; however I found a fresh umbilical hernia and a tumor mass in the left lower abdomen which seemed firm and made me suspicious of a new growth. We had consultation by a competent surgeon, and "There was a small umbilical hernia with a mass in the region of the left rectus which in his opinion was hemorrhage in the area of the sheath of the left rectus." He recommended ice packs and rest in bed for three days. At this time the employee's wife exercised the New York State free choice of physicians and took her husband to another physician. February 7 the second physician gave his opinion, a confirmation of my original idea that there was a fresh hernia and an older condition which was probably a new growth. The first mistake in this case was made at this time in that we did not have x-rays to determine if there was any chance of showing the presence of new growth in the abdomen. A positive picture would have been of some value, while a negative would have been of no value. February 10 the employee's wife requested her Doctor to get busy and operate on the hernia and find out what was really wrong with her husband. February 11, under general anesthetic, DR. O'L. and I repaired Mr. M.'s umbilical hernia and found a cancer about the size of an orange attached to his large intestine near the splenic flexture. This was very vascular and it was not possible to take a biopsy without incurring a great deal of hemorrhagic danger. Even though there had been no

obstruction at this stage I believe it would have been a good procedure to have done an illio-sigmoidostomy at this time regardless of the possibility that the insurer would have had a possible death claim. The attending surgeon gave us his opinion that the hernia was a fresh traumatic affair developing within a couple of weeks, while the cancerous growth was probably of at least two months' duration. My last yearly examination of this man was January 18, 1936, and the attending physician had examined him for a possible appendicitis attack November 1, 1936, at which examinations no evidence of the new growth was detected. Following the operation, repairing the hernia, there was an infection in the hernial incision which necessitated his staying in the hospital until April 3, 1937. His resistance was very low and he was very emaciated at the time he left the hospital. He was given liver injections at home to try to build him up. The employee's family and attending physician had not started treatment for the growth at this time and when it was suggested did not see fit to proceed with treatment other than supportive treatment. June 12, 1937, the attending physician gave us a State Form 4C, which stated that in his opinion the disability due to the hernia had been entirely covered. June 21 the welfare superintendent, in charge of compensation, and the employee's wife and I attended the compensation hearing and asked that the case be closed. The compensation Judge directed us to continue paying compensation and instructed the employee's wife to take her husband to Memorial Hospital in New York City. I arranged for Mr. M. to go to New York June 26, and there the Doctors' opinions confirmed our original findings and they referred him to Dr. A. F. Hocker, at the Cornwall Cancer Clinic, for deep x-ray therapy treatment. He was given 15 treatments and developed some intestinal obstruction signs along with a diminution in the size of the mass. He gradually vomited all his intake, and October 14 the surgeon again operated, doing an illio-sigmoidostomy, bypassing the annular cancer mass at the splenic flexion of the sigmoid. Following this operation the patient did not improve, and he died of obstruction October 21. This history diverged a great distance from the hernia subject, but it was still recorded in the court as a compensation case—with a great possibility that we would pay something for the aggravation of a pre-existing condition clause.

IN PRESENTING our case to the court we offered two definite conclusions from the facts: (1) that the lesion of the transverse colon was a carcinoma, and (2) that the reported trauma bore no etological relation whatsoever to the lesion. Further to establish any relation of trauma to the development of a tumor, there are five accepted criteria which must be established:

1. The authenticity and adequate severity of the trauma.
2. Previous integrity of the wounded part.
3. A reasonable time relation between the trauma and the appearance of an established tumor.
4. Continuity of pathological changes or symptoms in the part and the appearance of a tumor.
5. Microscopical proof of the existence of a tumor.

In this instance the time relation alone completely disproves any possible relation between the trauma and the tumor of the bowel demonstrated 16 days after injury. It has been established by authorities in the cancer research field that the development of large bowel cancer requires from 10 to 12 months up to three years. It is more than probable that the duration of the carcinoma in this instance, as judged by the size which it had obtained, was one or two years. Although microscopic sections were not obtained from this tumor, the sum total of evidence seems definitely to establish it as having been a carcinoma. The question of the relation of trauma to the subsequent course and outcome of the case is hard to settle.

Dr. Ewing, authority on cancer, states that "When a patient with symptoms of abdominal disease receives a blow in the epigastrium and rapidly succumbs to carcinoma of an abdominal organ it is reasonable to assume that the trauma hastened the progress of an existing tumor."

In this accident however, as in relation of trauma to etiology, it seems essential to confirm the authenticity and adequate severity of the injury, and a reasonable time relation. If the authenticity of the injury is accepted it would seem that several facts cast considerable doubt on the quoted injury as a competent cause in hastening the progress of this tumor.

1. The location of the blow was below and to the left of the umbilicus, which would place any direct violence away from the site of the tumor as demonstrated at the subsequent operation.
2. That the injury was not severe was shown by the fact that on immediate examination there were no external signs of injury, such as abrasions, bruises, ecchymosis, etc., and no signs of peritoneal irritation.
3. The deceased was able to work seven days after the injury before reporting for medical examination. This would rule out any such sequela of injury, edema, or actual tissue disruption.
4. At operation, 16 days after injury, no late signs of such hemorrhage or other injury were apparent.
5. The patient did not rapidly succumb to any obvious increased rapidity in growth of the tumor. When seen five months after injury he was still in comparatively good general condition, being able to ride to New York City, a distance of 58 miles, and returning in an automobile, with no ill effect, considering the size of and obstruction caused by the tumor, and no treatment had been given.

It is entirely possible that the hernia developed due to the combined effect of a pre-existing mass of tumor plus the increased intra-abdominal pressure produced at the moment of injury. Using these arguments in court we were able to close the death claim by agreeing to pay all costs, and his two-thirds wages had been paid up to the time of his death.

At the present time all traumatic hernias are repaired with fascial repair, where that operation is at all possible. Each case is being placed at special light work after recovery from his injury, and we do not employ for heavy work a man with an enlarged ring. Many of the above cases are old-time employees, and our new employees are carefully checked for dilated rings, and taught how to lift before they start to work. An average of 3.2 hernial repairs per year on 1000 employees is not a high record, but we hope to keep this down by a very careful selection of men and more thorough training of employees, as well as a more careful placing of men in jobs suited to their physical condition.

Industrial Medicine and the Manufacturer

DONALD M. SHAFER, M.D.,
Director of Survey,
Committee on Healthful Working Conditions,
National Association of Manufacturers

MANY people, especially industrial physicians, have wondered just what form the National Association of Manufacturers' interest in industrial medicine is taking. This is, I believe, quite understandable, for the value of the N. A. M.'s work to those now busy in the field will be determined largely by the surety — and, at the same time, the open-mindedness — with which the subject is approached.

Some tentative answers to the questions that may arise can be given, and may be helpful at this time.

Early in 1937, a survey of the American people by the personal interview method was carried out by an independent research organization. The purpose of this survey was to ascertain what the "man on the street" thought about industry. Among many other opinions voiced by this cross-sectional group, 47% felt that industry could best improve future conditions by providing increased benefits for labor. Another significant fact found was that this same group of people were of the opinion that the manufacturers and industrialists have done the most for the country in the past 10 years. In general, the typical attitude of the people was that industry has done the most for the country in the past and that industry could do even more in the future.

These findings, and the desire for improvement of the country's conditions in all fields led the N. A. M. to organize its Committee on Healthful Working Conditions. The Association felt that an active and continuous program by the Committee for the improvement of industrial working conditions would be a desirable method of benefiting the worker. It is quite obvious that when workmen are better able to do their jobs everyone profits, either directly or indirectly — and the whole national picture is improved as a result.

The Committee felt from the outset that its members as business men would best be able to judge the practicality of any proposed standards and at the same time believed that the technical knowledge necessary to establish and maintain healthful working conditions could best be supplied by industrial physicians. For this reason an Advisory Committee was formed, the membership of which was published in an earlier issue of INDUSTRIAL MEDICINE.* These two committees decided that the major components of healthful working conditions for factories are as follows:

1. Plant Medical Services;
2. Sanitation;
3. Lighting;
4. Heating and Ventilating;
5. Safety.

Certainly, good practices in the above fields would mean good living conditions for the employee at his work and would be a long step in the right direction. As a method of treating these various subjects, the Committee recommended that an inquiry into the "minimum standards" in these fields should be the basis for study, and, in addition, they felt that a review of the actual applications of the standards by industrial units throughout the country, especially such practices as may exceed the minimum requirements, would be the next logical step.

THE principle of establishing minimum standards for subjects such as these is by no means new, as we all know. Most of the large industrial concerns have, in their own laboratories and their own practices, employed some yardstick of this sort. Technical and educational groups such as the American College of Surgeons, the American Standards Association, various large insurance companies, and the National Safety Council, as well as various research societies, including the American Society of Heating and Ventilating Engineers, the Illuminating Engineering Society, and the Air Hygiene Foundation, have employed this method for quite some time as a basis of comparison and as a means for setting up acceptable procedures. The N. A. M. felt, however, that a wider dissemination of this knowledge was desirable, particularly to the smaller industries of this country.

This point I would like to amplify somewhat. A program leading to the establishment of healthful working conditions depends on five men. First, it depends on the worker. His attitude towards his environment and his cooperation with health and safety measures play a considerable part in the success of any program. In addition, his health is the final criterion of the environment in which he is working. He is the "proof of the pudding," so to speak. The second man is the doctor. His knowledge and his observation of the plant and of the worker brings to light good or bad working conditions. He is rather like a monitor in the environment, and by his professional ability and evaluation of the hazards is a guide to proper adjustment

* Written especially for INDUSTRIAL MEDICINE.

* Vol. 7, No. 3, (March, 1938), page 123.

of the surroundings. In addition to this, he cooperates with, or is himself, a research man, who is the next person responsible. The research man determines how and when the environment may be prejudicial, and also how an unhealthy factor may be removed or modified. Following him is the engineer who brings the practical method and apparatus of control to the environment and makes it function. The remaining man is the employer whose executive ability and financial assistance are required to put the whole scheme into action. Each one of these five men is essential in creating good working conditions.

It is my feeling that in all too many instances the employer has not had the knowledge obtained by the technical and professional men of the five-man team made available to him. He has his business to run; his time is taken up by it. What opportunity does he have to keep up with the researches of technical men? How can he follow the articles in the medical journals? The doctor realizes the time-consuming task of keeping up with the reports on new procedures and apparatus in his own field. Many an employer does not realize what an adequate medical service and the establishment of good working conditions can do for his men, his plant, and himself, or he feels that he cannot afford such a service. For these reasons the work of the N. A. M. Committee is concerned, first of all, with coordination. By coordination I mean the gathering of the essential facts from the scientific side of industrial hygiene and their preparation so that the industrialist can see what they mean and what they can do for his factory.

As Dr. Heiser in his article in Industrial Medicine* stated, the second phase following coordination is "selling" the advantages of healthful working conditions to the man who can initiate their establishment, i. e., the employer. The N. A. M. is admirably fitted to do this; being itself made up of many employers, it knows their problems, and knows how to reach them. However, at present the coordination activity is the major interest of the Committee. The entire value of the endeavor may be lost by a precipitate rush into "selling" before the coordination is adequate. Following is a rough outline of the way we are studying the components of working conditions. This will undoubtedly be altered and supplemented and is by no means rigid or all-inclusive.

In the study of the types of medical service available to industry we have made subdivisions as follows:

1. Company.
   a. Minimum medical services.
   b. Adequate medical services.
   c. Extended medical services.
2. Mutual Benefit Associations.
   a. Company financed and managed.
   b. Company and association financed and managed.
   c. Association financed and managed.
3. Union Medical Services.
4. Private Industrial Medical Services.
5. Private Group Medical Services.

The definition of "minimum," "adequate," and "extended" medical services is of course excellently set forth by the American College of Surgeons.* The procedures and merits of these medical services are being studied with a view to their cost, efficiency, the degree of preventive medical activity, and the size of industrial unit they best serve.

In endeavoring to find the best standards of sanitation in industry, I feel that perhaps the most comprehensive work is that of the American Standards Association. Little or no literature is available on the question of the actual usage of good sanitation by industry. This may be partially due to the varying interpretations applied to the word "sanitation." To some authors it includes heating, lighting, and toxic dust control; others limit it to plumbing and sanitary fixtures. At this point may I say that I would welcome any information concerning the extent of usage of minimum-standard or above-standard installations in an industrial unit.

In the field of industrial lighting I have found great activity and consciousness of the part good lighting plays in healthful working conditions. The Illuminating Engineering Society, through its Committee on Industrial and School Lighting, has conducted numerous studies of the conditions in specific industries and a nation-wide survey of the actual practices of industry as a whole. This work on the problem of glare is a genuine contribution to industrial health.

The subject of heating and ventilating has many ramifications. Within this subject there properly fall the air-borne ordinary and industrial diseases. The Air Hygiene Foundation has been very helpful to us in this study. In the analysis of temperature, humidity, and air motion effects on the workman the researches of the American Association of Heating and Ventilating Engineers and the Harvard Fatigue Laboratory have been of great value.

The safety factors of an industrial working condition have of course been adequately and continuously brought to the attention of the employer for a long period of time by the National Safety Council.

These comments on the various agencies that have assisted our study are undoubtedly rather dry reading to the industrial physician. However, to a large percentage of manufacturers, especially the smaller ones, they represent knowledge of which he knows little or nothing; which he can use greatly to his advantage; and which should be brought to him in a manner he can understand.

Another point I have not mentioned and which is of definite monetary value to both the worker and the employer is the fact that ordinary everyday non-industrial disease costs them both more than any other single factor. Without letting it

* Vol. 7, No. 9, (September, 1938), page 541.

* See "Blue Book of Industry," Industrial Medicine, Vol. 7, No. 3, (March, 1938), page 131.

detract from the fight against industrial disease or injury, this fact could well be the keystone of a program such as I have outlined. A program which makes healthful working conditions available to all manufacturers cannot help but benefit everyone concerned — the workman, the private physician and his nurse, the industrial nurses and doctors, the employer, and consequently the community at large.

The American Association

—of Industrial Physicians and Surgeons, 1939 Annual Meeting—

THE Twenty-Fourth Annual Meeting of the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS, with the AMERICAN CONFERENCE ON OCCUPATIONAL DISEASES AND INDUSTRIAL HYGIENE, will be held on June 5, 6, 7, and 8, 1939, at Hotel Statler, Cleveland, Ohio. The Program Committee, under the chairmanship of G. F. SYKES, M.D., (842 East 79th St., Cleveland), includes M. H. CASTLE, M.D.; H. L. DAVIS, M.D.; D. L. VAN DUZEN, M.D., and R. C. ENGEL, M.D., Cleveland.

Chicago Society

THE CHICAGO SOCIETY OF INDUSTRIAL MEDICINE AND SURGERY is holding its annual meeting on Monday, November 21, 1938, at 8:30 P.M. in the West Room on the 22nd floor of the Medical and Dental Arts Building, 185 North Wabash Avenue, Chicago.

The Program under the Chairmanship of DR. EMILE C. DUVAL, presents DR. FREMONT A. CHANDLER on the subject of "Blood Circulation of Bone." The discussion will be opened by DR. GEORGE L. APFELBACH and DR. RAYMOND W. MCNEALY. All members of the profession are invited.

Georgia Industrial Surgeons

THE Georgia Industrial Surgeons' Association held its first annual meeting at Sea Island Beach, near Brunswick, November 5, 1938. Officers of the Association are: DR. C. F. HOLTON, Savannah, President; DR. ROBERT L. RHODES, Augusta, Vice-President; DR. JOHN W. SIMMONS, Brunswick, Secretary-Treasurer.

Executive Health

THE department of industrial medicine of Northwestern University Medical School sponsored a dinner meeting at the Blackstone Hotel November 15, at which the theme of discussion was "Health of the Business Executive—A Disturbing Industrial Problem." Dr. Robert M. Daley, medical director, Equitable Life Assurance Society, New York, and Dr. Edgar Van Nuys Allen, associate professor of medicine, Graduate School of Medicine, University of Minnesota, Rochester-Minneapolis, were the speakers.

Noise in Industry

ROBERT LINDAHL
Celotex Company, Chicago

THE subject of this talk being "Noise in Industry," it might be well to start by defining just what we mean by noise. A definition that seems to fit most cases is that noise is any unwanted sound. For example, you might pay $5 to have heard Caruso at 8:00 o'clock in the evening, but at 3:00 o'clock the next morning, if you were trying to sleep and you heard him singing in the next apartment, that would be noise to you. So it isn't the quality of the tone, it is whether you want to hear it.

We are living in a constant sea of sound. We can shut out light, smell, or taste, but we can never completely eliminate sound.

The reason industry is becoming interested is because of the possible effect on individuals in regard to physical well-being, accuracy, labor turnover, attitude toward employer or personnel relationships, health, efficiency, etc.

What Noise Does

WE ORIGINALLY had little laboratory data. There was very little to be had 10 years ago. Dr. Laird, of Colgate University, initiated certain investigations. He experimented on human beings to see what their reaction was to noise under pitch differences and with different noise levels; and he also worked with rats. In the latter experiments, he had two groups of rats, one the "city" rats, and the other the "country" rats. The city group was to be exposed to the same amount of noise as encountered in the city, and the country rats were to be in a very quiet location.

In analyzing the subject of noise and its effects upon animals and upon human individuals, Dr. Laird found that you experience a fear reaction when you are exposed to a loud noise. First, there is an increased tension of the voluntary muscles; second, a lessened activity of the involuntary muscles in the digestive tract; third, an increased pulse rate; fourth, an increase in blood pressure; fifth, a diminished secretion of saliva and digestive juices; and sixth, a feeling of apprehension and restlessness.

In attempting to learn exactly what happens to an individual doing a certain task in a certain way under different noise levels, he had typists type exactly the same piece over and over under conditions of noise at 50 decibels and 35 decibels. It was discovered, in general, that the typists consumed about 19% more energy in a noisy room and had an increase of about 5% in output in the quiet room.

Now, it is possible that you can do as much work in a noisy room as in a quiet room, but you probably have to consume more energy in doing so. Your automobile can run along at 30 miles an

* Presented at the Twenty-Third Annual Meeting of the American Association of Industrial Physicians and Surgeons, and Midwest Conference on Occupational Diseases, Chicago, June 6-9, 1938.

hour on level ground and at 30 miles uphill, but you have to have more power to push that machine at 30 uphill than you do on the level ground. The human individual seems to react in the same way in ability to concentrate and to work under noisy conditions. If you are going to keep the same level of production, you have to use more energy.

Laboratory tests on mental efficiency were made and it was found, for example, that by reducing a complex noise from 50 to 40 decibels, there was a 30% increase in speed in persons multiplying three-place numbers by three-place numbers. The same degree of accuracy prevailed under all conditions.

Comments on Fatigue from Noise

IT IS difficult to arrive at the exact effects of noise, because people are not very well versed in the language required to describe their sensations. So I have copied a few quotations from persons who have used quieting treatment and have compared conditions before and after certain locations were quieted.

For example, Dr. Herman N. Bundesen, Health Commissioner of Chicago, whom you all know, said in one of his talks: "In one of our stenographic offices in the Board of Health, it was found that many of the girls were frequently absent from work. An effort was made to lessen the amount of noise in the room by installing acoustical materials on the walls and ceiling to absorb the noise. It was surprising how quickly this change cut down the absences, improved the efficiency of the girls, and reduced errors. Another important effect was the improvement in the temperaments of the girls. They became more pleasant as the irritation of noise was removed. From this it can be seen that the proper use of acoustical material to prevent unnecessary noise will go far to prevent the wear and tear of the nerves of the workers."

Another quotation, from the head of a stenographic department employing a number of girls: "This lessening of noise has resulted in pronounced absence of the fatigue which was formerly noticeable toward the end of the day. The girls do not have to make initial exertion to combat the tiring effect of constant noise, and we feel that increased efficiency naturally follows as the result of decreased fatigue and relieved nerves."

A life insurance company: "Our employees now finish the day less tired and with fewer cases of headache. The work is handled much more quickly and accurately and the same number of employees have practically eliminated night work, which was the rule before."

In other words, it seems apparent that there is an effect of noise upon workers; that they respond better and work better under conditions of relative quiet.

In order to sell management upon the fact that noise is costly and harmful, it is sometimes necessary to sell them on the idea of other factors than employee health and employee general welfare and personnel relations. I want to quote from a few other references regarding the increase in efficiency and output.

In moving the assembly department of a temperature regulator company from next to a noisy boiler shop to a quiet location, rejections were lowered from 75% to 7% and increased output resulted, going from 80 to 110 units in the same space of time.

In another department, removing the noise of a ventilating fan increased the output 12%.

In one of a telegraph company's offices, it was found that there was a 42% reduction in errors in the operating room after the noise level in the room had been lowered from 50 to 35 decibels; and this made a 3% reduction in the cost of the message.

The best example of the unbiased study of the effect of noise on workers is that given by an independent insurance organization, the Aetna Life. Insurance Company. Aetna was going to build a new building and the officials wanted to get complete data upon whether or not noise control should be considered in the construction of this building. Their investigation carried them over a period of two and a half years. In certain departments they had very accurate production records of the individuals because they were on a piecework bonus system. For one year they kept the records of these individuals without any quieting treatment. After installing quieting treatment, they kept the records for another year, and then as a control, went back to unquieted conditions for six months. These conditions all applied to the same workers on which the data was based.

They found that in a variety of machine operations there was an increase in efficiency of about 8.8%. Because of this, and because they found that the workers were better satisfied, they thought it worth while to quiet the entire new building.

It is difficult to find a similar case where efficiency records have been kept for as long a period under controlled conditions.

I shall not attempt to discuss the effect of noise on hearing, as to whether it raises your threshold level, because I do not feel qualified to discuss it before a group of physicians. Sometime we may have more data on this, but at present I do not feel there is enough that would be of interest to you as industrial physicians and surgeons, nor do I know of any cases where that has been a factor in the compensation settlements.

IT IS difficult to obtain averages of the effects of noise on individuals, because they vary so much and some are not sure what they hear. Not all people respond the same way, and that seems to be especially true in their hearing. I remember one case where we had put some acoustical treatment in a church in Baltimore. The pastor asked one of the older members of the congregation whether he thought any improvement had resulted. The old man replied: "Well, you are always talking about the Bible and I know it by heart. I'll be darned if I know whether I am hearing you or whether I know it all myself."

You do get cases like that where people do not know whether the noise has affected them or just what they are hearing, and it is diffcult to get them to express themselves.

With trained workers in the field of sound, such as they have at the Bell Telephone Laboratories, it is possible to get fairly accurate data with a large group. It is difficult, however, to go into any ordinary plant or office and get good data from a small group of workers.

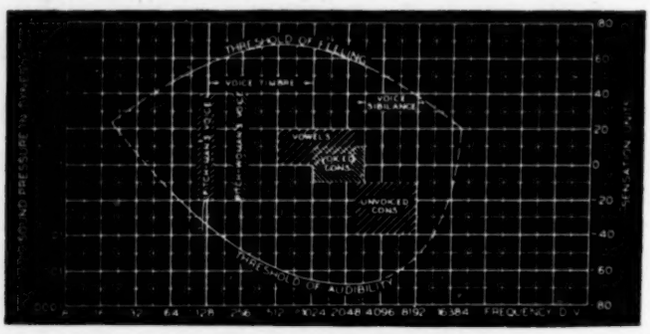
I might summarize the effect of noise on individuals by saying that its reduction does affect efficiency in output, and shows up in reduced absences, in a lessened employee turnover, and in better personnel relations. This is from the experiences of many firms.

I am sorry I do not have much data on noise control in factories. Most of the data I have given has been on offices. Possibly in a few years, we will have more information.

How Sound Is Measured

I WANT to take a few minutes now to go into the elementary physics of sound and measurement to give you an idea of the engineering difficulties encountered in attempting to lower noise levels.

(First Illustration): This audiogram is of a normal individual and is probably familiar to many of you. The area within the lines indicates the region in which the human ear receives sound sensation. We have pitch along the abscissa or horizontal axis and we have intensity energy along the ordinate or vertical axis.

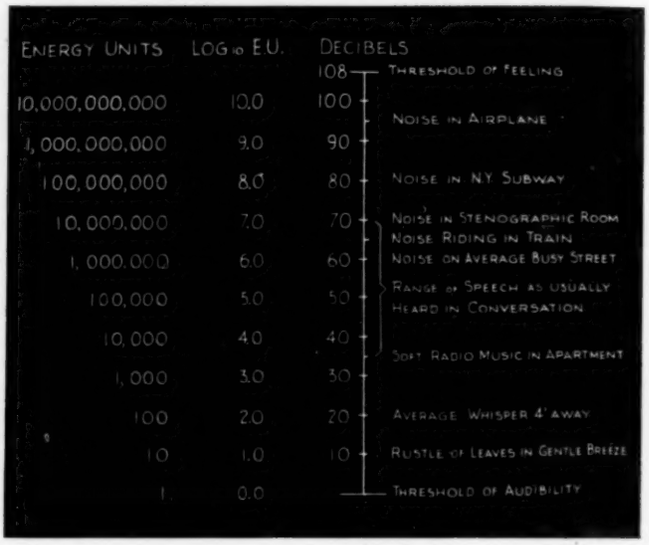


Hearing Diagram
Under the new standardization "decibels" (db) are used in place of "sensation units"

The human ear will perceive sounds roughly from 20 to about 20,000 cycles, and there is a range in intensity from about zero to 130 decibels. You will notice the ear is most sensitive somewhere around a thousand cycles.

(Second Illustration): It is difficult to explain the relationship between sound energy and loudness, but I am going to attempt to do it in a few words. In this column on the left on the chart we have energy units. Imagine that you are standing out in an open field and you see a man blowing a whistle some distance from you. You know he is creating sound energy but you just can't hear him, so we will say that is zero loudness and one energy unit. For us, that is the threshold of audibility.

Imagine now you have 10 men blowing whistles, and you will be able to hear that sound. We will call that one unit of loudness. In order to double the loudness of that sound, it will be necessary not to have 20 whistles blowing, but to have 100 whistles. If you wish to have the sound three times as loud, it will be necessary to have 1,000 whistles or 1,000 energy units; and if you wish to have it four times as loud, you will have to have 10,000 energy units or 10,000 whistles. In other words, loudness increases as the logarithm of the physical energy.



Decibel Scale
The above diagram represents a "Noise Thermometer," and gives the relationship between physical energy units and sound intensity level

In going from one whistle to 10 whistles, you will have gone through approximately 10 loudness steps, and in going from 10 whistles to 100, you will have gone through another 10 loudness steps, which are called decibels.

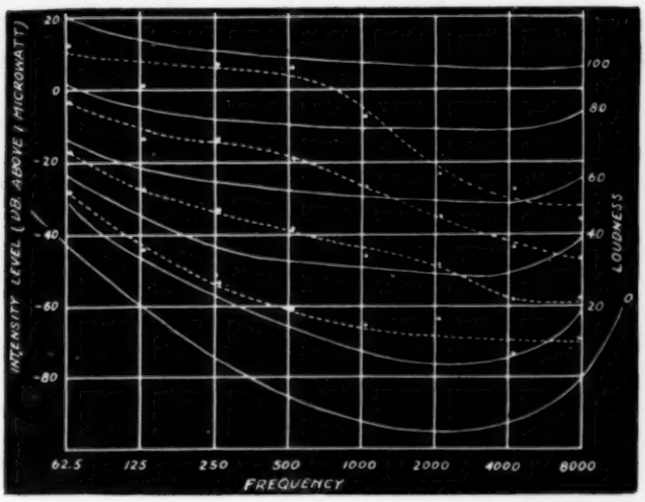
The rough definition of a decibel is the least perceptible difference in loudness which the human ear can perceive. For example, you can tell the difference between 50 and 51 decibels but not between 50 and 50.5 decibels. That definition is not strictly accurate because a decibel is really a ratio of the power difference of two sounds, and the one decibel difference indicates a difference in sound energy by an increase of 26% and an increase of 10 decibels indicates an increase in energy of 10 times.

We have a scale of loudness on our decibel scale, and you can see where common sounds normally fit. The level of conversation may be from 35 to 70. When you get up to the threshold of feeling—where you actually do not hear, but feel the sound, as you might standing by an airplane motor—you will be up to 100 to 130 decibels, depending on the particular scale being used. Noise levels above 40 db. may be considered as high enough to warrant consideration for quieting.

The noise level in this room, which I measured a few moments ago, was approximately 50 to 55 decibels. This is a little too high for good hearing. The speaker has to raise his voicee about 10 decibels above that level in order for you to hear his voice clearly instead of being conscious of the extraneous noise in the room.

You will see how difficult it will be to reduce loudness levels in a room where you have a certain amount of sound. If you take away half of the sound energy, you don't have half the loudness —you have only decreased it by three decibels. If you have 10 machines operating in a factory and you silence half of them, you will have only cut down the loudness in the room by three steps, barely perceptible steps. So, in order to get a decrease that is really worth while you have to cut down your sound energy to possibly 30% or less than the original.

(Third Illustration): This graph is intended to present the manner in which sounds of different pitch appear to the ear in terms of loudness by showing equal loudness contours. The previous scale was not concerned with pitch, but as you



Loudness Level and Annoyance Contours
The above chart shows the relationship between equal loudness levels (solid lines) and equal annoyance levels (dotted lines) as compared to sound intensity level. For example, a sound of 20 db. above threshold at 125 cycles sounds equally as loud as a sound at 500 cycles which is about 25 decibels lower in intensity level. High frequency sounds are much more annoying than low frequency sounds of the same intensity level

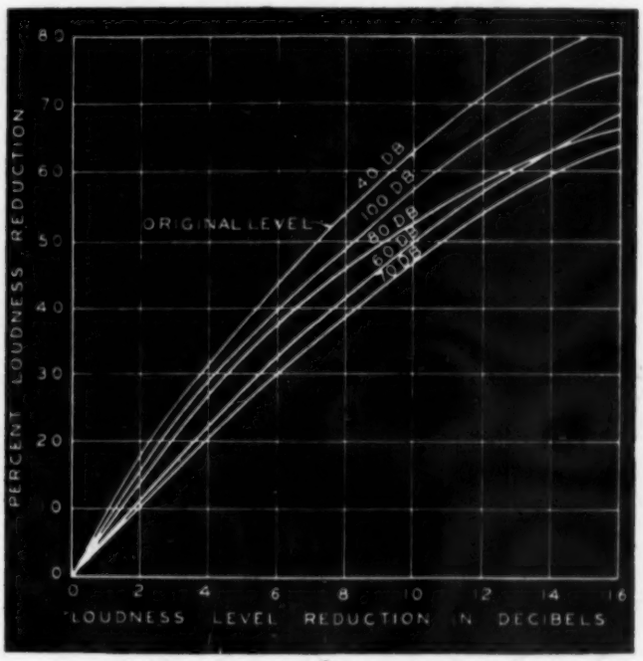
were shown on the first illustration, the ear is most sensitive in the neighborhood of 1,000 to 2,000 cycles. The loudness of a sound at 2,000 cycles may appear to the ear the same as the loudness of a sound at 125 cycles which is about 35 decibels higher in sound intensity.

Unfortunately, the ear does not record annoyance, which is primarily what we are concerned with, in exactly the same ratio as it does loudness. So we have the relationship of sound energy, or physical energy, to the intensity level in decibels and then to the loudness level as the ear perceives it, and then we are trying to translate it into annoyance level.

The dotted lines of the graph represent equal annoyance contours, so that any point along this line represents the same amount of annoyance. You see that they roughly follow the equal loudness curves, but when we get into the higher pitches they cross and come below.

In other words, sounds at high pitches are more annoying to the human ear than the sounds of lower pitches of approximately the same loudness. Sounds at high pitches may be very annoying whereas sounds of low pitches of the same intensity level may not even be heard. I merely want to bring in the relationship, not to confuse you, but to show you some of the technicalities in trying to relieve the annoyance due to noise and in trying to reduce the noise levels in rooms where such high levels do exist.

(Fourth Illustration): The ear does not always translate a loudness level reduction of different intensities of sound in the same manner. We have here a chart showing the loudness level reduction in decibels as against the per cent of loudness reduction which the individual feels has been



Loudness vs. Percent Reduction
Lowering a 70 db. sound 8 db. gives an impression of a 38% reduction in loudness, while lowering a 40 db. sound 8 db. gives an impression of a 54% reduction in loudness

achieved. You will see here that an 8 decibel reduction of a 70 decibel sound, produces in the average individual the feeling that loudness had been reduced about 38%; whereas, for an 8 decibel reduction of a 40 decibel sound, the average individual would say the loudness had been reduced 53 or 54%.

So, we have the further complication that sounds of different loudness levels do not appear to be equally reduced in loudness with the same reduction in intensity or loudness level.

I want to give a little demonstration, merely to illustrate the difference in loudness levels and of sounds at different pitches. Fortunately, within the last few years, we have had a standardization of noise measurement. You may know that the standard of common acceptance for the threshold of hearing is 10^{-16} watts per square centimeter. You will realize how little energy that amounts to

when you realize that one of these electric bulbs, burning about 40 watts, consumes many billion times more energy than that which exists at the threshold of hearing.

I am going to start with the sound of 250 cycles. [Sounds were generated for the audience to hear, by the special apparatus present.] The loudness of that sound is 62 decibels. I am now going to 500 cycles, and the loudness of that sound is 72 decibels; 1,000 cycles with a loudness of 80 decibels; 2,000 cycles with a loudness of 74 decibels; and 4,000 cycles with a loudness of 92 decibels.

I don't know whether you will all agree, but I think most of you will, that the high pitched sounds are more annoying to you than the lower pitched sounds. The meter I have here is a standardized sound level meter, which has a standard threshold and three different networks for measuring sound and weighting it in the same way as the human ear does at loudness levels of approximately 40 decibels, 70 decibels, and 100 decibels. It takes into account the characteristics of the human ear as I just attempted to explain them on these illustrations. Formerly, in order to make sound measurements like this you would have to carry around three trunks full of instruments. They were called portable because they had a handle, but it was a job getting them around. Now with an instrument weighing about 35 pounds you can take all the noise measurements you want and get almost all the data you need. If you wished to separate the data in the octave bands of sound, you would need in addition an acoustical filter, which is pretty heavy, so we left it at home.

How to Overcome Effect of Noise

THE next question is: How shall we overcome effect of noise in the average factory or in the average office? The best method is the direct one of eliminating the noise at its source. We now have quiet machinery, which it was not possible to obtain before. We have noiseless typewriters, quiet fans, quiet motors. Gears are made so that instead of having steel against steel, we have composition against steel, and a quieter sound results.

Less vibration exists when you get into quiet machinery and very often, the designer finds in trying to achieve quietness, he has also achieved efficiency. For example, the Rolls Royce was always a more efficient machine than the old Ford. The Rolls was much quieter and operated more efficiently. That is the way with a great many machines.

Very often by cutting down vibration you may also cut down a harmful effect upon the worker. I remember in an addressograph room of an insurance company in Indianapolis, the girls were complaining of the noise in the room. Looking it over, I noticed that the machine which they used to punch the plates was vibrating so much that one of the girls had actually to chase the keys. There was about a half-inch amplitude. With a properly designed machine, not only would it have been more efficient but there would also have been less strain on the worker.

Unfortunately, we can not always isolate the noise at the source. You can not easily quiet a 50-ton punch press. You can not always quiet a lathe. There is always a certain amount of noise, and so we often have to go to an indirect way of accomplishing the end.

Isolate Noisy Machines:

One of the ways is to isolate these noisy machines or other types of noise producers in one particular area and then build sound insulating partitions so that other workers are not affected. It is also possible to put vibration isolaters on the machinery so the vibration does not carry through into the building structure. When you are riding along in an automobile, you have what you call a cruising speed. You go along very, very smoothly. If you go over a bumpy road, you may happen to hit the resonance point and your car seems to be bouncing and chattering. It is the same way with machinery. With properly designed springs and supports you can have a cruising speed in the machinery and it can operate smoothly and efficiently. With the wrong kind of supports or merely setting the machinery on the floor, you may have a bumpy road effect, with the machine bouncing around and sending noise and vibration into the building and shaking itself to pieces.

Indirect Method of Quieting:

The next indirect way of quieting is by the use of quieting materials. In a room such as this, the plaster and the glass absorb about 3% of the sound that strikes them. The carpet absorbs about 30 to 40%. When you go into the average factory or into the average office where you have hard non-absorbing material, like glass, plaster, wood, and concrete, the noise which is created rapidly spreads to all parts of the room, is reflected back and forth, and is not absorbed but continues to reverberate anywhere from five to 10 seconds.

By having sound absorbing materials in the room, this sound immediately hits the sound absorbent materials and so much energy is absorbed at each impact that the sounds do not last very long and can not build up to a higher amplitude.

You know that most men like to sing in the bathroom. The reason is that in the bath, everyone thinks he is a Caruso. You have the hard tile walls amplifying your voice and you think you have tremendous power. You never hear of anyone singing in a clothes closet. All the clothes are absorbing the energy from your voice. You feel as though the sound is being pulled out of your mouth. The result isn't pleasing and your tendency probably would be to whisper rather than to shout. Thus you have a psychological effect with sound absorbent treatment.

There are certain limits which you can achieve by the use of quieting materials in rooms where noise exists. In general, you can not secure more than a six to 10 decibel reduction. This may not seem very great when you figure the range on the noise scale from zero to 130 decibels, but as shown on the chart before, this may result in a decrease in loudness according to the average worker of anywhere from 30 to 70%.

I am sorry that I do not have more data as applied to factories, as I said before. I have had to translate some of the findings we have had in offices to factory work, but I think I can summarize by saying that noise is definitely harmful in industry; that the workers use more energy under noisy conditions; that it lessens their accuracy when they are performing painstaking tasks; that it lessens their output; and that it does affect the employee's morale and health.

Noise can be eliminated or reduced in most places where it is economical to do so. That does not mean in a room where you have one low-salaried man working that it is worth while trying to reduce the noise level. But where you have many people working in one room, and there is a definite effect on their health and morale, it is usually possible to do something about it by decreasing the noise level.

Discussion:

DR. CAREY P. MC CORD (Detroit): So far as several of us recall, this is the first paper on noise that has been presented before any of the outstanding associations interested in industrial hygiene. At once it reflects the alertness of the Program Committee in bringing this topic to our attention. Also, it reflects the alertness of the speaker to have such excellent material to bring to us to fill the need for information on this subject.

There is a very interesting thing about noise as an industrial exposure. Most exposures begin when the workman leaves his work bench or work place. The workman is exposed to silicious dust; something may be gained from time to time by eliminating long hours; at once, the exposure is lessened. But, when a workman leaves a very noisy place, he is apt to go to his home or some other situation where there is likely to be as much noise but of a different sort. Instead of working with a machine in the factory, he may go home and have his nights made hideous by radios, street cars, automobile horns, to say nothing of the woman who starts her tap dancing on the floor above at 11:45.

Noise is always with us and noise leads to a fairly definite series of diseases to which we have applied, in a recent paper, the term "echeoses." There is always some offence at creating new terminology, but if you are interested in a term that will cover all noise disturbances, the term "echeoses" will probably apply.

Of all echeoses, deafness from noise is the outstanding one, and this is not a figment of the imagination. There are many industries where noise is such that not less than 50% of all workers, even though they have an average age of not more than 30, may be shown to have lost up to 50% of their hearing capacity.

One of the worst industries in the whole country, so far as noise deafness is concerned, is that of drop forge works. It is not so much a matter of noise there, but also motion imparted to the air by the terrific impact of the forge hammer. Recently I was talking with an employment manager who had a drop forge division and I asked him how he picked his drop forge men and how he knew they were experienced. He said: "It is easy. If they put their hands up to their ears and ask: 'What did you say?' I am apt to employ them at once because I know they are experienced drop forge men. But if they can hear as well as I can I know they have had practically no experience, because all drop forge men with a few years experience are apt to be deaf due to their employment."

In many types of work, the most practical means for eliminating some of the ill effects of noise is by the direct insertion in the ear of noise deflectors. It's not an entirely admirable thing. Some of the deflectors are made of plastic material which may lead to aural eczema, etc., but that is better than becoming deaf or partially deaf. A number of these ear deflectors are now on the market which are made of plastic material and various types of rubber. Some are good and some bad. Perhaps, after all, the best protection may come from the ordinary mixing of vaseline with plugs of cotton and inserting those in the ears.

I would like to talk about many other echeoses, but time does not permit. May I close with this idea: Twenty-five years ago, young doctors were seen to run around, trying to impress the public by having in each hip pocket an obstetrical forcep. That was their badge of honor. That day has passed, and now no useful purpose will be served by anything such as that. But a useful purpose will be served if you young doctors and you young engineers may be seen going into industry with a noise meter in one hand and an audiometer in the other, getting at some of the very genuine problems connected with noise and noise deafness.

DR. E. M. MIKKELSEN: Can ideal acoustics be predicted in a building, or is it on an empirical basis?

Mr. Lindahl: At the present stage it is possible to design an auditorium like this so that almost perfect acoustical conditions will result. In other words, we can tell that the average person will be able to understand the speech and that the average person will like music played in that room, but when it comes to reducing the noise level, that is a different proposition.

Acoustical quieting or treatment consists of treating a room so the audience can hear a speaker clearly, but noise quieting consists of putting in as much treatment in a room as is necessary to cut down as far as possible the harmful noise—anything above a level of 40 decibels.

An Aviator and His Ears

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WHEN prehistoric man emerged from the muddy waters of an antediluvian swamp, he underwent, for the first time, stresses incident to adapting himself to a new environment. The pituitary gland is thought to have been at that time a piece of optical apparatus recessed in the vertex of the skull, and vertical vision, probably useful in submarine activities, became no longer necessary. It is presumed that it thereafter became rudimentary and its function was supplanted by the better development of the elevator group of eye muscles. Regardless of whether other aquatic appendages were similarly caused to become rudimentary, the fact remains that after reaching dry land, because of absence of protective mechanisms, man thereafter became peculiarly prone to pay the penalty of sinus infection whenever he has attempted, in swimming and diving, to assume the role of an amphibian.

It is probable that man, with increasing mental development, became attracted to the sounds of heavier-than-air, prehistoric flying monsters, and, with commendable ambition, began his long and bitter but persistent attempt to fly.

When man realized a yearning to fly, it was necessary for him, in an adaptation from a terrestrial to a celestial environment, to suffer stresses of poor anatomical compensation for a new and unusual existence.

As is well known, we lead a precarious and

marginal existence on the floor of a great ocean of oxygen. Our reserve supply of oxygen is so limited that under conditions of complete oxygen deprivation, we can live but a short time. It may be confidently stated that in final summation, every death is due to anoxemia. In experiences of stratospheric flying, humans have, for short periods only, been able to emerge from the surface of an atmospheric sea and momentarily catch a glimpse of the great azure blue, mysterious realm of the unexplored sky beyond. Unfortunately man, even on the floor of this sea, is constantly confronted by the "muddying of the waters," so to speak, as the result of which he is subjected to stresses so manifold that it is a source of unending wonder that any survive. When one goes beyond the limitations of his natural environment, he finds it necessary to lean very heavily upon mechanical, protective devices. It is possible for him, if unprotected, to change altitude so rapidly that he may become unconscious from lack of oxygen or from effects of accelebration, the victim of nitrogen bubbles in his blood, the classical "bends" of the diver.

Certain medical standards have been set up as to hearing acuity among pilots. One official regulatory agency requires that a pilot demonstrate ability to hear the unaccentuated whispered voice at 20 feet. In another standard, the same test is applied at a distance of eight feet. The clicking coin test and the ticking watch test are applied, the usual requirement being ability to hear the sounds at a distance ranging from 12 to 40 inches. In one set of regulations it is recommended that audiometer tests be applied, and if a hearing loss of 15% is demonstrated the pilot should be disqualified.

In former days of commercial flying, in open cockpit type of airplanes, pilot alertness to hear a skipping motor and to atune one's hearing to the humming of wires was necessary. Humming wires, so long as emitting clearly heard sounds, gave indication that proper flying speed was being maintained. It came to be a pronouncement among pilots that if the humming of the wires subsided materially, one was about to approach a degree of stalling flight—the last step before falling off into a spin. The proprioceptive apparatus enabled one to recognize a draft of wind on a cheek and to thus recognize a skid or a slip; if not corrected, flying speed would be lost and a spin would be inevitable.

As pay loads became increased, equipment became heavier and horsepower was tremendously increased. Flying with visual contact was no longer economical in the maintenance of schedules. It became necessary to develop so-called blind-flying instruments with the use of which it was possible to proceed through weather which, in the light of previous experiences, would not only have been impossible but extremely hazardous.

The modern airliner is built with a closed pilot compartment. In flying such heavy, fast equipment, where visibility is frequently and completely obscured, it is not possible for one to rely on his sensations as to spatial orientation. In fact one's mental impressions become so perverted in blind flying that, without the aid of instrument, a crash would surely occur.

In the beginning of aviation medical standards, a great deal of emphasis was placed upon the necessity for normal sense of balance. This presupposed the existence of a normal, healthy vestibular apparatus. While such requirements are still in effect, the purpose, now, is to demonstrate that the pilot is in good general health, in respect to his labyrinths and to brain stem. This is in order to be assured that severe, spontaneous vertigo or syncopal attacks will not likely occur. In instrument flying the pilot has to disregard his mental impressions as to spatial orientation, he must rely entirely upon his instruments. In the event of motor or other structural failure, vibration and instrument board indications of various kinds will inform the pilot much more quickly than it is possible for him to sense the situation through his proprioceptive apparatus or special sense organs.

AIR LINE pilots average 33 years in age. Each has flown in excess of 5,000 solo hours. A very large number of solo hours have been accomplished entirely by instrument flying under conditions of complete obscuration of visibility. Here, the pilot relies implicitly upon his dual panel of blind flying instruments. Sense of hearing is most important in the field of radio communication. If the pilot has complete loss of hearing in one ear without impairment of his sense of balance, he has, for the purpose of hearing through a radio ear phone, normal hearing. He has normal hearing except that he cannot directionalize sound. In the field of air line piloting, the ability to directionalize sound is of little importance and constitutes a defect which is in no wise as serious as it would be in case of a railway employee who, working in a freight yard, would be unable to directionalize sound or to sense the direction of approach of uncontrolled, moving, rolling stock.

Carefully conducted audiometer studies among a large group of airline pilots, many of whom have been flying for 15 to 20 years, reveal striking absence of serious occupational deafness. One would normally expect to encounter hearing losses among a group who, in former years, had been exposed in open cockpit airplanes, to prolonged, violent motor and other noises. The average air conduction loss was 5.5%; the average bone conduction 9.7%.

Radio operators constitute a vital element in successful blind flying. In order to pass through an area of low visibility, the pilot must be kept informed of weather conditions ahead in order that he may, at all times, and with a wide margin of safety as to reserve gasoline supply, land at an airport where open weather is reported. If the radio operator, through faulty ability to hear, could not maintain ground communication with the airplane in the sky, a definite hazard would result. The radio operators of Eastern Air Lines, Inc., were subjected to studies as to hearing acuity. The average age of the radio operator is 28 years. The average air conduction loss among radio operators is 2.7%. Bone conduction loss average is 11.7%. The ears of the radio operator are highly trained. The radio operator works continuously for eight hours. He uses a so-called split headphone set

through which, in one ear, he hears the spoken voice, radio code signals in the other ear, and the spoken voice or radio signals from a loud speaker, nearby. This task is accomplished in a highly satisfactory manner. Consequently, upon being tested with the single sound of the various cycles in an audiometer test, the task is a simple one for the radio operator. It is noted that his responses are much more rapid than the responses of the average layman whose work does not particularly train him to recognize telephonic signals of various kinds.

It is said that the air line pilot is subjected to more occupational ear stresses than any other type of worker. He is constantly being subjected to the effects of changes in altitude. As one ascends, atmospheric air in the middle ear expands. When the pressure within the ear becomes great enough it escapes through the Eustachian tubes. If, perchance, there is an acute congestion of the nasal passages, contained air may be unable to escape from the middle ear. Consequently, bulging of the ear drum and, in rare cases, a rupture, may occur. In descending from high altitudes, there is a contraction of air within the middle ear. If the Eustachian tubes are not patent, various degrees of retraction of the ear drums will follow.

Passengers are also subjected to the effects of changing altitude. Each is given chewing gum in order to stimulate the frequency of the act of swallowing. A sleeping passenger is more prone to troublesome ear symptoms upon descending because, while sleeping, he does not swallow frequently.

Airlines have promulgated and rigidly enforce strict regulations which provide that the ship shall descend at a rate not exceeding 400 feet per minute. At this rate of descent, if one indulges frequently in the act of swallowing, little discomfort will result.

On some of the major airlines, the flight attendant, either a hostess or steward, routinely instructs all passengers who appear to have acute colds, as to protective measures; and, in some cases, supplies them with inhalers, the constricting effects of the drugs therefrom giving them sufficient shrinkage to afford relief and protection.

The incidence of serious ear trouble among pilots and passengers, because of the protective technique of flight in present airline operations, is fairly rare as to occurrence. In the extremes of flying as represented by military operations and by aeroacrobatics, there is a definite ear hazard. This subject has been given comprehensive thought by Harry G. Armstrong, M.D., Captain, Medical Corps, United States Army and J. W. Heim, Ph.D., in an article entitled "The Effect of Flight on the Middle Ear,"* Captain Armstrong has brought into medical nomenclature the term "Aero-otitis media." This article may be read with benefit by those who are especially interested in this subject.

Passengers, unless actually ill, may, by observing ordinary precautions, travel by airplane without discomfort. Children seem to be less prone to unpleasant ear symptoms than adults.

In summary, it may be stated that, from the view point of an air line medical department, the pilot who can hear the unaccentuated whispered voice through a standard audiometer microphone circuit with amplification such as on the Maico Audiometer, and who has no serious impairment of hearing in the frequency of the conversational range, is regarded, so far as hearing acuity is concerned, to be fully qualified for air line piloting duties.

* Published in *J.A.M.A.*, August 7, 1937.

Age Incidence of Hernia

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SEVERAL forces are at work to extend the period of years that the employee spends in industry. In addition to the general increase in longevity, industry is witnessing a gradual entrenchment of the seniority system. Unfortunately, this brings the old employee no respite from the disabilities and infirmities of age.

It seems probable that the relation of age to industrial health hazards will merit increasing attention. The present study is concerned with the incidence of inguinal hernia in a group who have been given routine industrial examinations. Some of these were pre-employment examinations, and some were among men already employed. None was employed in organizations where pre-employment examinations had previously been made to eliminate men with hernia, and the group therefore shows no selection on this basis.

A "hernia" is taken here to mean an inguinal hernia, direct or indirect, with a palpable impulse

TABLE I
INCIDENCE OF HERNIA AT VARIOUS AGES

Age Group	No. Examined	No. with Hernias	Percent with Hernias
16-20	320	5	1.5
21-30	1388	63	4.5
31-40	1046	113	10.8
41-50	650	105	16.1
51-60	205	48	23.4
61-70	63	26	41.2
71-80	8	5	62.5

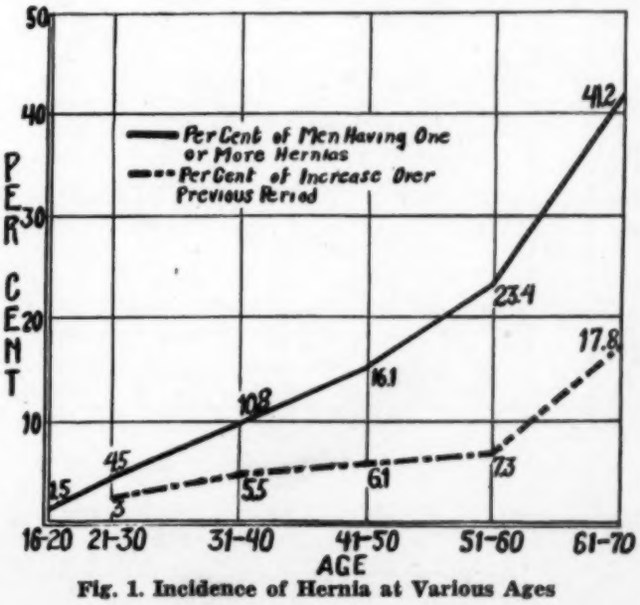

Fig. 1. Incidence of Hernia at Various Ages

at the external ring. "Potential hernia" or "loose rings" with no impulse are not included.

The group represents 54 different employers in 23 types of business, and is, therefore, probably a fair cross-section of industry at large.

A total of 3680 men were examined. Of these, 365, or 9.9%, showed inguinal hernia. This percentage includes those with repaired hernias, whether the operation was successful or not. In the youngest age group, only one man in each 66 is ruptured. This rate rises rapidly, as shown in Table I. The eighth decade is not plotted because of the small number examined; however, it is, at least, suggestive that the percentage (62.5) if plotted, could have made a smooth prolongation of the upward curve.

The dotted line in Fig. 1 is plotted by subtracting, from the percentage in each decade, the percent in the previous decade. The purpose of this is to point out that rising curve of total hernias does not merely represent an accumulation. There are actually more new hernias developing in each succeeding decade.

Of the 365 ruptured men, 86, or 23.6%, had bilateral rupture. The age incidence of these is shown in Table II. This is likewise plotted in Fig. 2, omitting the eighth decade.

All in all, the 365 herniated persons had a total of 451 hernias. Sixty-seven of these hernias or about 15%, occurring in 63 persons, had had a surgical repair. In the 67 repairs, there had been 17 recurrences (25.4%).

Comment

THE most frequently quoted theory of the genesis of hernia is that it occurs where some failure of normal obliteration of the peritoneal canal is present. This may very well be correct, but if this factor was the principal one it is only logical to believe that the incidence of hernia should be greatest in the early years of life. Instead, it would appear that the greatest incidence of hernia parallels the degenerative processes of age. Of these processes, the most important would seem to be the loss of muscular and fascial strength. It is generally appreciated that this feature of age begins to show itself early. Muscular efficiency as measured by competitive standards begins to fade in the third decade and proceeds to do so, at first slowly, then at constantly accelerating rate suggestive of Fig. 1. I believe this offers adequate explanation for the observation that the older a man gets, the more apt he is to become ruptured.

In the younger decades it is apparent that a man with one hernia is more apt to develop a new one than the man with none. This is most marked in the third and fourth decades where the incidence of new hernias is twice as great in men who have one already. Later in life this tendency diminishes, and during the sixth and seventh decades it would appear that there is even less chance of a man developing a hernia if he already has one. The absence of an apparent explanation for this offers a tempting field for conjecture. What the real reason for this is, I do not know.

The surgeon operated on only one hernia in seven; 85% are still untouched. It is impossible to say how many are injected because this procedure leaves little external trace. The 25.4% recurrences seems high. In view of the much better percentages obtained by many surgeons and clinics, this emphasizes the discouraging results that many operators must have gotten. This factor, added to occurrence of double hernia, makes the employee with the recent repair by an unknown operator still a poor risk. Thus a man between 20 and 30 years old with a recent well-repaired single hernia, has a 25.4% chance of his hernia coming back, and a 12.6% chance of getting one on the other side within 10 years. Since recurrences usually appear early such a man would have nearly a 40% chance of getting a rupture, new or recurrent, within a decade.

TABLE II
INCIDENCE OF DOUBLE HERNIA

Age Group	No. Hernias	No. with Double Hernias	Percent with Double Hernias
16-20	5	0	0
21-30	63	6	8.8
31-40	113	25	21.6
41-50	105	30	28.6
51-60	48	15	31.2
61-70	26	8	30.8
71-80	5	2	40

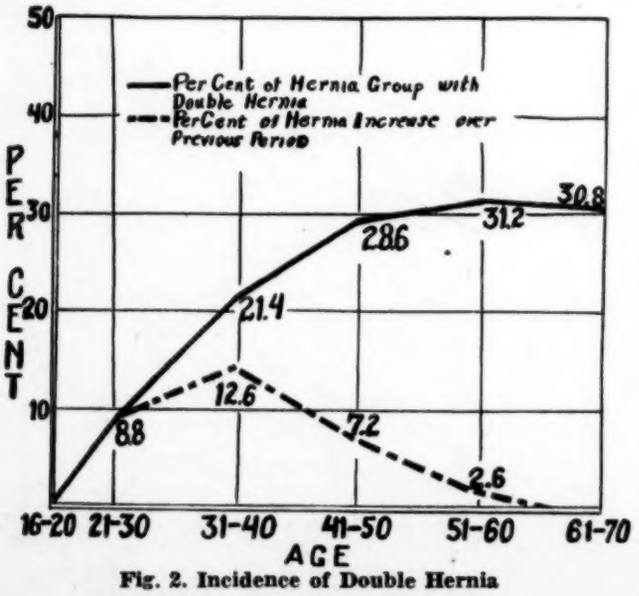

Fig. 2. Incidence of Double Hernia

Conclusions

ONE in 10 of the men studied were ruptured.

2. At 16 to 20 years of age, one man in 66 is ruptured; at 61 to 70, 40 men out of a hundred are ruptured.

3. Rupture is a disease of age, its appearance paralleling the gradual weakening of the supporting tissues.

4. The older the employee, the more apt he is to develop a rupture.

5. One hernial repair in four in this group developed a recurrence.

6. In the first five decades a new hernia occurs more commonly in those who have one already, but in the sixth and seventh decades this ceases to be true.

7. In the older age groups, about one ruptured man in three will have a double rupture.

Note on Breathing

WE HEAR much these days of idiosyncrasies to poisonous gases.* From my respiratory studies on 400 men I found that many individuals even at rest breathe from 50 to 100% more air than their fellow workers of the same age, height and weight. In workers with a large vital capacity (measured by the maximum amount of air that can be expelled in one breath) the bellows or lungs have less work to do in the performance of a given task. The worker whose breathing is inefficient often inhales twice as much air (thus doubling the gas exposure). In my opinion those individuals are not more susceptible to toxic gases, but have a greater exposure due to their being what I have termed inefficient breathers.

Cutaneous Hazards in the Fur Industry

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In Collaboration with
Howard T. Behrman, M.D.,
New York City

"STEEL Worker Slips into Flaming Crucible!" "Vat Overflows; Three Men Scalded to Death!" We are all familiar with headlines such as these which appear from time to time in the daily papers, describing various occupational accidents. The public reads them with mingled pity and horror; pity for the individual victim, and horror that accidents can still happen in industry in spite of the additional precautions taken each year. The facts themselves are sensational without the aid of reportorial eloquence. Occasionally, even the story of an occupational disease will make the front page, so long as the press finds some human interest involved. Columns devoted to the women afflicted with radium poisoning from painting watch dials attested to this. Slow death combined with monetary awards created the almost perfect news story.

Diseases of the skin acquired by furriers in the natural course of their labors do not however have the benefits of publicity. We will never read over the morning coffee headlines such as these: "Dyer Victim of Paraphenylenediamine!" "Fur Stretcher Stricken with Severe Dermatitis!" Or "Furrier Scratches Rash; Secondary Infection Develops!" There is nothing intrinsically dramatic about irritated areas of the epidermis: they are capable of being cured. You cannot blame the public for showing no interest in an occupational disease which responds readily to medical treatment provided the patient cooperates with the physician. But, although the layman exhibits no curiosity about fur dermatitis, the problem is not without interest.

Since 1913 when Blaschko[1] first studied modern conditions in the fur industry resulting in occupational diseases, approximately three-score articles have been published on various phases of the subject. These articles range all the way from brief statements of case histories appearing in medical society bulletins[2] to a well documented thesis presented before the Faculty of the University at Bordeaux in partial fulfillment of the requirements for the degree of Doctor of Medicine by Naudillon[3] and an excellent Public Health Bulletin by Louis Schwartz.[4]

With fur prices dropping to a new low level, and the installment plan beckoning encouragement, this appealing commodity now comes well within the budget of women who long considered fur coats a prohibitive luxury. With the market thus expanding, more and more employees will be required to take the raw skins through the difficult processes which fit them for use, and with the increase in the actual numbers of furriers employed will come an increase in the number of cases of fur dermatitis observed by physicians.

THE hazards of preparing animal skins for the retail consumer actually begin with the threat of anthrax for the men who handle raw pelts fresh from infected animals. The skins, as they arrive at the dressers', are either raw and moist, or dried and powdery, depending on the type and size of the pelt. Large skins are usually moist, small ones, dry. If raw, they immediately undergo a process of blood-letting which is accomplished by exposing them for several hours to a current of heated water. Should they be dry and brittle, they must be revived by immersion in a salt, acid or formic solution. Just as the raw skins in their natural state may communicate an animal disease, so do the dried skins threaten health. For example, the skins prepared for shipment in China are coated with a rice powder preservative whose composition is highly irritating to the skin. A 17 year old boy among our patients, working as a moistener and stretcher of furs for six months, suffered from dermatitis of the hands, fingers and forearms. Patch tests to determine causation excluded the possibility of dye or tanning chemicals, but his skin showed positive reactions to dye powders, such as the Chinese wood used in the glazing operation. The excess of drying powders on all the skins of this type, no matter what their source, heightens sensitivity to allergic dermatitis and asthmatic conditions. The drum filled with moist sawdust which tosses about small pelts till they regain their pliability, also endangers the workers unless superior ventilation and forced drafts carry away the clouds of irritating dust.

When the raw skins, including the dried ones that have been softened by soaking, are passed through the flesh-stripping process, they are pulled to and fro over large, sharp fleshing knives to remove the fragments of muscle and fat still adhering to the skin. The constant friction involved wears the fingernails of the workers down below the quick, and where acid has been used for immersion, an erosive action may be noted. Some cases show reactions which are quite marked and exhibit tendencies to prolonged and chronic inflammatory changes. A peculiar onychia char-

* From an article on "Carbon Disulphide Poisoning," by Dr. Max Trumper, at the Ninth Annual Greater New York Safety Council, 1938.

acterized by dryness and a shiny appearance of the dorsal surface of the nail with recession of the free edge was pronounced in some individuals who had been active at the occupation for a long period of time. Numerous factors, such as prolonged moisture, or insufficient drying, exposure to acid and alkalies, and mechanical irritation all may combine in the production of the nail changes. The nail folds are frequently dark red in color, elevated, edematous, scaly and fissured. Frequently pus could be expressed from under the folds.

After the stripping of the skins, there follows the most important step in the preliminary preparation of furs: the tanning, the purpose of which is to treat the skins in such a manner that they will submit to dyeing and drying without further altering their degree of pliability. The tanning must be done either with a bath of gall, tannin or cachou, or by the application of oil—fish, grapeseed, or castor—or, as is usual, by soaking the skins in a 10% salt solution of alum, potassium, soda or ammonia combined with a small quantity of sulphuric acid. This salt-acid solution is capable of causing unfortunate cases of dermatitis unless the workers protect themselves by wearing rubber garments. A thorough rinsing with water is necessary to carry off the excess of the caustic base which might otherwise prove poisonous. Finally an active beating renders the skins still more supple and more capable of retaining the dye which is to follow. The beating is often done by hand and minute particles of hair and dust, unless carried away by special suction ventilation, will cause many cases of catarrh and asthma. Before the beating, the skins are usually stretched by being pinned to frames. The pricks of these pins often lead to the development of a skin condition from the tanning acids which otherwise might have been entirely innocuous.

The preliminary operations described above are requisite for all furs before styling. Dyeing, however, is not always essential. Those pelts whose natural color is highly valued are merely glazed, others, a little less beautiful, require only a brief coloring process to blend them with the perfect skins and thus augment their monetary value. By far the greatest number of skins, however, need to be worked over, sheared, dyed and glazed to imitate as closely as possible those pelts which are most in demand. Sable, the prince of all furs, may, upon close inspection, prove to be nothing more than the lowly martin, seals may have been born muskrats, and skunks unmask to opossums. Rabbit skins, with little or no intrinsic value, are so successfully disguised as seal, beaver, mole, etc., that the raising of rabbits has become a profitable industry. As soon as it becomes necessary to treat the hair of a skin, the furs must be "killed." A weak alkaline solution brushed over the hairs removes the fatty coating which would keep the dye from penetrating. Water removes the alkali, and a weak acid solution is used next for neutralization followed by further rinsing in clear water.

It is then indispensible to use a mordant, or cutting agent, particularly for deep colors. The use of this mordant leaves a salt capable of forming an insoluble and strongly adherent composition when combined with a coloring base on the hair itself. Otherwise the dyeing operation would be far from successful, the colors uneven, and not fast enough to resist either light or friction. The concentration of the mordants varies with the desired shade and they are largely taken from the metallic salts: bichromate of soda or of potassium, sulphate of iron or of copper, pyrolignite of iron, etc. By employing an alkaline mordant, these two processes may be merged into one. The application of the mordant, like that of the dyes, may be accomplished either by brush, or by immersion. The latter is the least used.

Immersion in the mordant is followed by immersion in the dye; rinsing comes next with the customary stretching and exposure to a drying temperature. The widely used process of brushing on the mordant and then the dye requires expert manual labor. It is essential that the hard brush, dipped first in the mordant and then in the coloring matter should pass over the hairs alone without injuring the roots or the skin itself. The workers engaged in killing, mordanting and dyeing are all exposed to irritating chemicals and must wear rubber gloves, aprons and boots to guard against the development of a sensitivity to the tools of their trade.

The dyes that are employed may be of mineral, vegetable or synthetic nature.

Among the mineral dyes are lead acetate, ammonium sulphide and potassium permanganate. These dyes, while entirely successful so far as color is concerned, are both too expensive and not without danger to the hair of the fur, and are therefore not in favor with the fur industry. The vegetable dyes are those containing tannin which are used in conjunction with iron salts to produce greyish to black shades. Nutgalls, sumac, chestnut and oak supply the tannin. Other vegetable dyes are obtained from logwood, yellow wood, quercitrin, Brazil wood or red wood, cutch, tumeric stem, etc. The vegetable dyes are used mainly for the most expensive furs like caracul, black Persian lamb and broadtail. They rarely, if ever, cause dermatitis among workers or wearers of the furs.

FOR good results at minimum cost, the fur industry employs almost exclusively artificial coloration. The most popular artificial dyes, created in the domain of organic chemistry, are the analine and diamine bases, particulary the phenylindiamins. Paraphenylenediamine is the usual type. Trade names such as Ursol and Nako momentarily conceal the chemical constituents of the dye in use. The chief characteristic of salts from these bases is that they oxidize very easily in the air and especially in the presence of oxygenated water. Dyeing with these colors therefore means that, after mordanting, a solution of the selected base is applied to the hairs alone, and oxygenated water is added. By varying the proportion of the water, it is possible to vary the intensity of oxidization, which results in a whole range of shades

and tints. Color modulations include yellows, greens, beige, grays, browns, etc. These colors are noted for being fast, resistant to air and light, to rubbing and to washing. What with the simplicity of the operation and the proved color fastness, there is no question as to the reason for their popularity. And yet, the use of these dyes is not without inconvenience to a percentage of the men who work with them, while the action of the organism is frequently disagreeable.

Paraphenylenediamine itself [C_6H_4 $(NH_2)_2$] is not as irritating as its intermediate products. Paraphenylene diamine, a scaly substance, soluble in water, alcohol and ether, becomes transformed through complete oxidation into a quinone known as Bendrowski's base, which is an insoluble product, closely adherent to hair and harmless to the skin. But before oxidation is complete, and here is where the danger lies, it turns into an intermediate substance, quinondichlorodiamine, which is a positive skin irritant. In its dry form, or in solution with alcohol, neither meta- nor paraphenylenediamine applied to the skin causes more than a slight erythema. The toxic products are the dimethyl and diethyl derivatives. Hanzlik[5] reports in his study of this group that except for acetylcholin, there are no substances more poisonous when applied to skin which has suffered an abrasion. The harmful actions of the paraphenylenediamines is direct and chemical, not anaphylactoid.

Not only are the furriers subject to the dermatitis resulting from partially oxidized dyes, but when the crystals are not thoroughly washed out of the fur, the wearer may be a victim of the same skin condition due to continued oxidation after the garment has actually been manufactured. Cutters and piecers of fur pelts are often affected by the flying particles of hair, especially in the crevices surrounding the nose, the angles of the mouth, the corners of the eyes and the creases of the neck. Moisture makes the dust adhere to these specific points, and this very moisture is essential for the liberation of the oxidation products in sufficient quantities to cause ill effects. Quinone, when applied to the skin in a moistened condition proves to be immediately irritating, but this is no doubt just one of the poisonous stages through which paraphenylenediamine passes during its soluble state. Louis Schwartz includes this dye in his list[6] of "specific irritants" which cause dermatitis only among certan hypersensitive individuals who suffer from an allergy to this particular chemical compound. It is perfectly true that only a small percentage of workers suffer ill effects. When factory studies are made, however, it is likely that those men who found themselves sensitive to the various chemicals involved were no longer working at that occupation. Should an employee discover a dermatitis during the first few weeks of his new employment, he is apt to blame the job and promptly leave it. In this way, the workers adjust themselves, accepting employment only where their health remains normal. This is especially true, of course, of sufferers from skin ailments which are obvious to the layman, rather than for lung or bronchial conditions which are fully or partially developed before they are recognized. Consequently, statistics covering factory personnel tell us very little about the percentage of persons sensitive to dyes, since they deal with those workers who have been in the industry for years just because they felt no ill effects from their occupation. The New York State Department of Labor reports that in 1936 there were only 53 compensation claims in the fur industry, of which only 33 were allowed. This leads us to suspect that furriers who develop dermatitis after many years at their respective jobs, may be suffering from non-occupational skin diseases, as many of the cases referred to us have so proved. In fact, 16 out of 50 cases referred to us proved to be suffering from non-occupational skin diseases, all the way from simple fungus infections to the outward manifestations of bone tuberculosis.

PATCH tests utilizing the products with which the patient comes in contact, are good clues in determining causation, if they are positive. When they are negative, they actually mean nothing, because they indicate nothing about the patient's previous history of sensitivity, nor are they prophetic of the patient's future sensitivity under differing conditions. In giving a patch test, the seat of the test is chosen as near the eruption as possible and on an unaffected area of skin in an attempt to approximate the same conditions. The reason for this choice is that certain sections of the cutaneous surface, such as the palms of the hands, are known to be less liable to external irritations. A solution of the suspected chemical is applied to this chosen area and kept there for a specific period to see whether irritation will result. It is essential to learn the exact amount of concentration of the chemical to be tested, and the time required for a normal skin to react to the agent for purposes of comparison. It is often advisable to use an unaffected worker as a control.

To assure a similar condition of skin, three types of perspiration may be employed as part of the patch test; the patient's at the time of the test, a synthetic preparation having an alkaline reaction, and a synthetic preparation having an acid reaction. Since perspiration is affected by diet, any of these conditions might have been the one existant at the time of irritation. Obviously, the chemical contents of perspiration influence its ability to act as a solvent on external irritants, or as a neutralizer. An alkali exposed to an acid type will be neutralized and thus afford protection, but should the perspiration be alkaline also, a non-poisonous solution may be so strengthened that the unconscious augmentation of the alkaline factor will actually create the irritation. This is equally true when acid meets acid. When these three possibilities have been included in the tests, the theory is that the physician has covered all the variants possible in the physical set up of the worker that might have rendered him subject to unpleasant chemical reactions with the agents above mentioned. Should the patch test prove

positive on one or all of those areas previously prepared with different type perspirations, the result is, of course, gratifying. But should the patient fail to react to the patch test, that does not conclusively prove that he was not sensitive to the chemical involved at a specific and perhaps limited time when the irritation was first noted. General health debility following illness may have sensitized the victim who may still have the dermatitis when the doctor sees him but not the weakness which originally caused him to contract the disease. Among our cases we have the example of a fur cutter, who was suspected of a sensitivity to dye, but in whom a simple sunburn probably caused the predisposition. Sunburns as well as the pricking of the skin with stretching pins—in fact, any thinning or weakening of the cornified layer of the epithelium, invites the entry of external irritants.

Out of 50 cases selected at random, seven were definitely predisposed by pricking their fingers, or tearing their skin on nails or wire brushes. These seven either actually remembered the scratch or abrasion at the time of its reception, or it was still apparent for the physician to see. Many others however, principally the fur nailers and sewers may have suffered scratches and pricks too slight for them to notice, but not too slight to act as inductive areas of the skin for the infiltration of poisonous substances. Even in the study of so specific a predisposition as skin breakage, it is impossible to judge accurately what percentage of workers were victimized in this fashion who normally would have been able to work for years, and in some of our cases, did, without suffering any ill effects from their occupation. One must not forget that a negative patch test can never truly reproduce actual working conditions, such as the purely external factors of heat, cold, sunlight, friction, etc.

When it is difficult to pigeon-hole the comparatively simple situation of external breaks in the skin for lack of accurate information, one can imagine the insurmountable difficulty of making out a specific case against allergy as a predisposition in the study of fur dermatitis. The field of speculation broadens perceptibly, until the burden of proof becomes positively evasive. Dr. Schwartz's definition of allergy as a "hypersensitivity in which an antibody is produced in the system in response to an external irritant acting as an antigen"[8] has the virtue of being concise. Unfortunately, the antibody cannot be discovered in all cases, and the antigen often proves as elusive as the intermediate dye oxidation products. Dermatitis in the fur industry is probably more often encouraged on the one hand, by changes in the simple defense mechanisms of the skin which lead to increased vulnerability, and on the other, by excessive or unusual amounts and concentrations of an external irritant. Nevertheless, when a certain individual whose general skin condition does not seem to have undergone any recent modification and whose general health and hygiene are beyond reproach, is the only one to contract dermatis of all the workers engaged in the same processes, working side by side with him and exposed to the same irritants, then this man obviously differs from his fellow creatures in the degree of hypersensitivity to specific stimuli.

Allergy is probably a familial, congenital predisposition but it also may be acquired over a space of time by continual exposure to various elements both through the cutaneous surfaces of the body and the respiratory system. This acquisition of hypersensitivity may work both ways. Eventual immunization may evolve after constant exposure to an irritating agent. Conversely, nitro compounds belong specifically to the group of sensitizers which, as the time element becomes elongated, lead to an allergic condition: they actually produce an antibody.

When allergy alone is the cause of dermatitis, there are three possibilities. Normally the skin irritation is slight, lasting only a few days or weeks and immunity follows; or a severe dermatitis will manifest itself just as soon as the individual becomes exposed to the irritant. No immunity will follow this latter type and the occupation responsible will have to be given up tc afford relief. The third and most unusual case is that of a man who after 20 years or so at one job without exhibiting any skin eruptions, suddenly finds himself sensitive to chemical agents which he has handled for years without difficulties of any nature. These men will obviously never develop an immunity.

AN ANALYSIS of the 50 patients referred to the author in private practice as suffering from industrial dermatoses, revealed the fact that 16 were suffering from cutaneous conditions not occupational in origin. The patients arrived at the office with conditions which had been described as occupational in cause by others. It is therefore clear that physicians who are not qualified dermatologists may have difficulty in diagnosing about 28% of the cases. Among the various non-occupational diseases which confused the diagnosis, were dermatitis secondary to a previously existant skin condition, seborrheic eczema, toxic erythema, folliculitis, urticaria, and especially fungus infections of the skin. Particularly noteworthy was the occurrence of dermatophytosis, the fungus infection of the skin which caused the most confusion in diagnosis. The differential diagnosis between dermatophytosis and occupational dermatitis depends upon the history, the location of the lesions, and their morphology, patch tests, the isolation of the fungi, and the effect of discontinuance of the work under suspicion of repeated exposure.

The trichophyton test should not be considered of final diagnostic value because of the great prevalence of the infection among all individuals. A positive test does not indicate whether or not the suspected occupational skin eruption from which the patient is suffering is due to a fungus infection but it merely indicates the presence of an allergic state almost universally due to the in-

fected feet. The claim by some individuals that the finding of a positive trichophyton test excludes the presence of an occupational dermatitis, we do not believe should be allowed. For instance, an occupational dermatitis of the hands and dermatophytosis of the feet may occur coincidentally and a positive biological reaction is misleading. Even a fungus infection may by chance be the result of a transmission of the disease from the pelts that are handled. It is also possible that among the cases regarded as non-occupational in this series, that some of the conditions, such as urticaria, folliculitis and toxic erythema may have been due to absorption of toxic agents as a result of the occupation, but after a careful examination of all the medical factors, this possibility was definitely disowned.

The patients under discussion were males in all but two cases, and they varied in age from 17 to 68 years. The great majority had worked in the fur industry for several years and one patient for a period of 45 years. Only one patient was less than 20 years old; the rest were in the third decade and older, up to the age of 68 years. In only one case was there a history of the patient having suffered from a previous attack of dermatitis due to his occupation in the fur industry. Certain factors predisposed to the development of the dermatitis. Six out of the 34 patients suffering from occupational dermatitis developed the eruption after a lowering of the resistance of the skin occasioned in one case by sunburn, in others by the prick of a nail or pin and by abrasions of the skin suffered during the scraping process. Heat, sweat, increased moisture and poor local hygiene all played a contributing part in the development or aggravation of the skin condition. Hot weather seemed to have a definitely bad effect on the patients.

There is no doubt but that seborrheic eczema and particularly a fungus infection of the feet, predispose the patient to occupational diseases by setting up a hypersensitivity of the skin. In those patients who developed lesions of the face, a previously existing irritation of the skin was conducive to the development of lesions at this site. The course of the condition was always of short duration except in those cases where the patient insisted upon continuing the work responsible for his disease, or where the skin was being irritated while doing other work in the presence of the active lesions. Undue prolongation was traced to poor hygiene in some cases, and in others to a hypersensitivity that developed into asthma in two cases. The patients considered in this study had been suffering from two weeks up to 16 months before they were referred to us for diagnosis and treatment. In all cases, the cure would have been accelerated, had the dermatitis been recognized immediately, and had not the application of irritating prescriptions meant to be remedies aggravated the original condition. Secondary dermatitis and occasionally secondary pyoderma were the unpleasant results of incorrect diagnosis followed by applications of irritating ointments. When the patients who had worked for some time in the fur industry suffered for the first time from a dermatitis, it was discovered on investigation that the composition or strength of the materials used had been changed before there were any cutaneous complaints. In almost every case, the lesions were confined to the fingers, hands and forearms, and only rarely was there involvement of other parts. Occasionally lesions developed secondarily on the trunk and lower extremities, regions far removed from the primary focus and where there had been no contact with the causative agent. Such efflorescences may be induced as a result of the absorption of biologic products being carried to sites whose resistance has been lowered because of poor constitutional states or a predisposed vulnerability of the skin in that area. Such secondary lesions tend to appear on the skin of the legs especially in the presence of faulty circulation as from vericose veins and where there are nutritional tissue changes. Vulnerable tissues with cell changes tend to attract circulating antibodies.

THE most common of all the cutaneous affections occuring among the fur workers was that of dermatitis. The condition should be recognized as an acute or chronic catarrh of the skin that results from contacts with irritants and sensitizing agents in the course of the patient's occupation. Therefore, in the prevention and treatment of the condition and the gradual elimination of this complication among furriers, the most important thing to bear in mind is the employment by the worker of measures to prevent prolonged and unnecessary contact with the offensive irritants. Could the owners of warehouses, dyeing establishments and manufacturing plants become aware of the role they play in maintaining the health of their employees by assuring them of proper hygienic conditions while they are at work, they would not have to undergo the loss of time and general inconvenience when one of their skilled workers is obliged to cease work because of an occupational disease. The workers themselves must assist in maintaining a high sanitary level. It has been observed that the man who does not bathe as frequently as his fellow men, suffers the most. By not washing his person frequently, the pilosebaceous follicles are allowed to become clogged with secondary involvement of the surrounding skin. Also, if a primary catarrhal condition is properly treated, secondary pyogenic infections and infiltrating lesions with a tendency to run a prolonged, resistant course would be obliterated.

Hypersensitivity would be less likely to develop when a man bathes often and launders his working clothes regularly. Of course, the ideal condition would enable the worker to use gloves for the finer manipulations which might be necessary in his work as well as when he is employed in a process obviously hazardous. Briefly, the prevention of the occurence of dermatoses among fur workers and the reduction of complications with prolonged courses would result if proper instructions were

given to the employees and if the employees acted upon these instructions in every detail. Eventually, fur factories will, when urged by the unions, see that their men have locker facilities so that they could change their clothes before and after working. Showers, wash basins in sufficient numbers, good bland soaps, preventive oils such as cotton seed, and emollients should be provided. An excellent industrial preventative soft soap containing lanoline would aid in removing the irritants and protect the skin from their harmful actions.

In the treatment of the dermatitis the usual topical applications are employed according to the pathology that is recognized. In a general way it may be stated that in the acute states, slightly astringent and bland wet dressings are used. With the subsidence of swelling, edema and vesiculation, lotions are better on the skin, and in the subacute stages, pastes are best. For thick, chronic and infiltrated lesions, ointments containing some form of tar and perhaps salicylic acid serve best. Antipruritics are frequently necessary and for severe pruritus especially with resistant chronic patches, x-ray therapy is most valuable. Ordinarily these measures are all that are necessary but other physical agents and local remedies may have to be administered to combat various local and internal symptoms, when the individual case so dictates.

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Lead Poisoning

—Section II: Fifty-one Fatal Cases of Suspected Lead Poisoning; and Forty-six of Non-Fatal Lead Poisoning; Diagnosis; Treatment; Summary—

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THE 10 non-industrial cases of lead poisoning among children reported here are concrete evidence that proper legislation should be passed which would prohibit the use of lead in toys, or lead paints on children's furniture or toys. Health officers should inform parents of the grave danger involved in exposing their children to lead in any form. These cases have often been thought of as acute, but they cannot be so classified because the period of exposure ranged from a few weeks to four years. However, the onset of acute symptoms is quite rapid and distinctive, and there is no doubt that these are clear-cut cases of chronic lead poisoning.

Case 1. A male child, age three years, nibbled paint from front porch railing for about one year (pica). The symptoms which developed about one week before death were refusal of food, vomiting, constipation, abdominal cramps, and convulsions. The autopsy revealed focal miliary hemorrhages in the cortex and medulla of the brain, edema and internal hydrocephalus. The cause of death was hemorrhagic encephalitis due to lead.

Case 2. A male child, age two years, chewed on painted plaster and lead putty for a few weeks (pica). The symptoms which developed two weeks before death were loss of appetite and convulsions. The clinical findings were a marked anemia, basophilic stippled erythrocytes, and a definite line of increased density (presumably lead) in the epiphyses of the long bones by x-ray. Previous to these findings the impression was that of an intracranial tumor. A surgical decompression was performed without success. The autopsy revealed an edema of the brain and thickening of the meninges. The cause of death was lead encephalitis.

Case 3. A female child, age two years, inhaled and swallowed lead-laden ashes. The mother used old storage battery cases as fuel in the kitchen stove for about one month. Within three weeks the following symptoms were exhibited: anorexia, diarrhea, drowsiness, vomiting, convulsions, ptosis of right eye lid, and flaccid legs. Death occurred one week after the symptoms developed. Each case and its sludge yielded approximately 190 grams of lead. The postmortem findings were bilateral bronchopneumonia, passive congestion of both kidneys, and a marked edema of the brain. The immediate cause of death was bronchopneumonia.

Case 4. A female child, age five years, had same exposure as that above. This child was a sister to the one in Case 3. The same history and comment as in that instance applies here, with the exception that an unsuccessful subtemporal decompression was performed and numerous stippled red cells were found.

Case 5. A male child, age four and a half years, had a history of pica for two years, gnawing on painted woodwork and eating dirt. The history revealed constipation for several months, vomiting one week, chronic convulsions in left arm and leg one day, and gums suggesting a lead line. The autopsy findings were hemorrhagic encephalitis, bi-lateral bronchopneumonia, and acute glomerulonephritis. The cause of death was hemorrhagic encephalitis due to lead.

Case 6. A female child, age seven years, had a history of pica for four years, chewing on "tin-foil" wrappers, painted articles, and eating dirt. She was ill for one week with loss of appetite, vomiting and abdominal cramps. An x-ray showed what appeared to be lead lines in the epiphyses of the long bones. An unsuccessful surgical decompression was done. An encephalitis and edema of the brain was found at the autopsy. The case was finally diagnosed as lead encephalitis.

Case 7. A female child, age three years, had a history of pica for about two years, gnawing on painted surfaces and eating dirt in the shop where her father painted signs. She was sick for one week with severe cramps, vomiting, diarrhea, and constipation. An acute follicular ileo-colitis, toxic encephalitis, and petechial hemorrhages in the medulla were found at autopsy. The cause of death was acute follicular ileo-colitis possibly due to lead.

Case 8. A male child, age two years, had a history of pica, gnawing paint from front porch railing for an unknown period of time. He was sick for one week. Many stippled red cells were found. The symptoms were loss of appetite, convulsions, and coma. The postmortem examination revealed an extensive edema of the brain and encephalitis, which was the cause of death.

Case 9. A male child, age two years, was exposed to lead dust and possibly lead fumes resulting from the burning of old storage battery cases as fuel. The child was having a convulsion when admitted to the hospital. The x-ray revealed the opaque line (apparently due to lead) in the epiphyses of the long bones. Severe convulsions occurred intermittently for 15 hours, followed by coma for one-half hour, and death. The microscopic examination of sections of the brain and the chemical analysis revealed that death was due to lead encephalitis.

Discussion: The nine cases reported here are characterized by a definite exposure to lead, typical symptoms of lead intoxication, and brain pathology ascribed to lead. The concentration in the bone is perhaps lower than might be expected. This is

probably due to relatively short periods of exposure and to the fact that the sternum and ribs were mostly cartilage. Children probably ingest more lead than adults in a given period of time because they are totally unaware of any hazard. This fact, rather than any special selectivity, probably accounts for the higher concentrations of lead in their soft tissues as compared to those in the adults.

ADDENDUM: The following case occurred too late to be included in Table IX and to be given the proper case number. For convenience it has been added here.

CASE R.D. A well-nourished four year old colored boy was brought to the hospital in convulsions by his mother June 29, 1938. The mother stated that she had used old storage battery cases as fuel during the past two years, and that the child had three convulsive seizures accompanied with gastro-intestinal upsets during the past year. Two intravenous injections, 10 cc. each, of a sterile 1% solution of di-sodium phosphate were administered as an emergency measure, but the child died the following morning.

Postmortem findings: brain very large (1390 grams), semi-firm, gray matter was gray-brown and congested, convolutions of cerebrum and cerebellum were flattened; cloudy swelling of the myocardium, liver, kidneys, and spleen; swelling and dusky red discoloration of the entire gastro-intestinal tract. The following concentrations of lead were found: liver 4.40, kidney 1.05, sternum and ribs 26.24 mgm. per 100 grams. The cause of death was an acute exacerbation of chronic lead poisoning with encephalopathic changes.

Twenty-One Chronic Industrial Cases

THE great majority of suspected lead poisonings are of a chronic industrial nature. They are chronic because absorption, deposition, and storage of lead in the tissues has been gradual but progressive over a relatively long period of time. When the concentration of lead in the tissues reaches a certain range, one or more symptoms of intoxication will usually develop in the majority of cases. The symptoms may be the direct effect of abnormal amounts of lead on the hitherto healthy tissue cells. In fatal cases of encephalitis and nephritis where there is a history of abnormal exposure to lead and the chemical analysis shows an abnormal concentration of lead in the tissues, the encephalitis and nephritis can be ascribed to lead with a reasonable degree of medical certainty. However, it is necessary to show that there was no evidence of the disease prior to the onset of a toxic episode of lead poisoning, and experience

TABLE IX

NINE CASES OF NON-INDUSTRIAL CHRONIC LEAD POISONING IN CHILDREN

Case No.	Exposure	Symptoms of Lead Intoxication	Concentration (mgm. Pb per 100 gm.) Liver	Kidney	Bone (Sternum and Ribs)
1	One year of pica	Very definite	6.60	3.10	
2[a]	Few weeks of pica	Very definite	6.00		6.00
3[b]	Ashes from battery cases for 1 month	Very definite	3.75	2.00	3.00
4[c]	Ashes from battery cases for 1 month	Very definite	3.74	2.89	4.00
5	Two years of pica	Very definite	2.03	0.93	5.70
6	Four years of pica	Very definite	1.28	0.99	1.91
7	Two years of pica	Somewhat indefinite	0.74	0.16	0.85
8[d]	Few weeks of pica	Very definite	1.89	0.30	9.16
9	Ashes from battery cases for about one year	Very definite	2.13	1.03	13.47
	Mean		3.13	1.43	5.38
	Probable error of mean*		±0.69	±0.39	±1.47

a—Lead in brain, 0.51 mgm. per 100 Grams.
b—Lead in intestines, 1.20 mgm. per 100 Grams
*No cases were omitted in this calculation.
c—Lead in intestines, 1.50 mgm. per 100 Grams
d—Lead in brain, 0.29 mgm. per 100 Grams

The observed symptoms were continuous twitching of the facial muscles, superfluous secretion of saliva, sucking the tongue, clenched fists, convulsions, coma, and a glassy stare of the eyes. The convulsions were not associated with a rise in temperature. X-rays revealed marked lead lines in the epiphyses of the long bones. Spinal fluid: 12 lymphocytes, 4 plus Pandy, and increased pressure. Blood: hemoglobin 60%, 3,800,000 red blood cells, color index 0.8, 16,900 white blood cells, 88 polymorphonuclears, 10 lymphocytes, 2 monocytes, and 10,000 normoblasts per 100,000. No basophilic stippled cells were seen by Wright's method. Blood drawn while the child was still living yielded 0.22 mgm. of lead, which amount is about seven times the "normal" and indicates lead intoxication.

reveals that cases such as these occur very infrequently among adults. Cases occurring most frequently are those in which (1) the immediate cause of death developed or was aggravated during lead intoxication, and (2) an acute attack of plumbism is precipitated by some acute infectious disease which develops a condition of acidosis within the body, such as influenza or pneumonia. The immediate cause of death in the following cases was not due to lead poisoning, but the clinical and chemical findings indicate that the victims were suffering from lead poisoning just prior to or at the time of death.

CASE 10. A man, age 56 years, worked as a barrel-scraper and clean-up man at a white lead company for 21 years, apparently inhaling abnormal concentrations of white lead dust. He had several attacks of lead colic during the last three years of life and was being treated for lead poisoning. The type of treatment was unknown. He was ill and unemployed for eight months prior to death. Symptoms: abdominal cramps (lead colic), loss of appetite, general weakness, and swelling of legs. Postmortem findings: arteriosclerotic kidneys, cysts and calculi in kidneys, concentric hypertrophy of heart, arteriosclerosis. The immediate cause of death was chronic nephritis. The chronic nephritis and arteriosclerosis could be ascribed to lead, but that is a matter of conjecture.

CASE 11. A man, age 32 years, was a painter for six years and worked until the very day of his death. Symptoms: loss of appetite, abdominal cramps, constipation, marked anemia. Postmortem findings: ulcers of the stomach. The cause of death was hemorrhage of the stomach. All of the symptoms could be due to the gastric ulcers and they could also be due to lead. The concentration of 0.4 mgm. of lead in the stomach shows that lead was entering by way of the gastro-intestinal tract on the day of death. Ulcers are caused by conditions other than abnormal amounts of lead. In this case it is a safe conjecture that the spreading of the lesions (if not originally caused by lead) was apparently accentuated by it. The abnormal concentrations in the other organs indicate that he was very likely suffering from a certain degree of lead intoxication.

CASE 12. A man, age 50 years, was employed at a lead plant for 15 years, melting and pouring solder. He had complained of pains in the back, chest, and abdomen, gradually became worse and died during the second week of a vacation. The possibility of silicosis originated from some source, perhaps based on pains in the chest in spite of the absence of an abnormal exposure to silica dusts. Postmortem findings: carcinoma of right kidney and right adrenal, carcinomatous lymphangitis of both lungs, metastasis of periaortic and peribronchial lymph nodes, myofibrosis cordis. The immediate cause of death was carcinoma of the right kidney. Silicosis was ruled out because of the absence of any abnormal exposure to silica dusts, because of the exceptionally low silica content of the lungs as compared to 50 other cases in which the writer has determined silica, and because a microscopic examination of lung sections showed no silicotic nodules or fibrosis in the opinion of a competent pathologist. The plant physician ruled out lead poisoning because no basophilic stippled cells were found, which fact may be very misleading in many cases. There was a high concentration of lead in the liver, and a marked deposition in the bone indicating storage over a long period of time, coinciding with the history. Lead in the lungs was not high, though above normal, possibly explained by a relatively complete absorption during the interval between the last exposure and death. In the absence of a family history of cancer and any toxic substance other than lead acting as a constant irritant, it is possible that lead was the causative agent of the malignancy if such condition developed during lead intoxication.

CASE 13. A female, age 20 years, was employed for eight months winding dyed silk thread on metallic spools by high-speed machinery. Several months previous to the above employment she was operated upon for appendicitis and ovarian cysts. Severe abdominal cramps came on while on the job. She was sick in the hospital for two weeks. Symptoms: abdominal cramps, bilateral wrist-drop, neuritis, constipation, violent and irrational. Postmortem findings: bronchopneumonia, hyperemia and edema of brain, lungs, liver, kidney, glomerulo-nephritis. She died of bronchopneumonia in a psychopathic hospital. The dyed silk and metallic spools which were said to have been used failed to reveal more than traces of lead, so the lead in the organs came from some unrevealed source.

CASE 14. A male, age 46 years, was employed in a paint factory for 18 years handling white lead and at times packing lead arsenate in the insecticide department. He quit work two months before death, with the following symptoms: abdominal cramps, extreme vomiting, and nephritis. There was a lead line on the margin of the gums, secondary anemia, and many stippled cells. Postmortem findings: bronchopneumonia, glomerulo-nephritis, perforated septum (due to arsenic). The exposure, symptoms, and chemical findings fit perfectly into the picture of lead poisoning but he was undoubtedly suffering from chronic arsenic poisoning at the same time. Information about the exposure to arsenic was revealed too late to make an analysis for it. Death was due to bronchopneumonia.

CASE 15. A male, 42 years of age, was employed in a paint factory 18 years cleaning and welding breaks in machinery used in mixing white lead. He had three attacks of "severe lead colic" in two and one-half years, abdominal cramps and constipation, and quit work four days before death. Postmortem findings: peritonitis, ruptured peptic ulcer. He died from peritonitis following a ruptured peptic ulcer. The abdominal cramps were possibly due to the ulcer, or to the effect of lead on the gut, or both. It is possible that the ulcer was caused by lead.

CASE 16. A male, age 27 years, scrapped and soldered auto radiators for seven years. He left work five weeks prior to death with abdominal cramps and constipation. There was a marked secondary anemia, hemoglobin 35%, and many

stippled cells. Postmotem findings: bronchopneumonia, nephritis, fibrinous plastic peritonitis. The immediate cause of death was bronchopneumonia. The chemical findings are most irregular. Exceptionally low figures for the liver and bone and a very high concentration in the kidney indicate a condition of extreme acidosis wherein deposited and stored lead had been largely liberated and excreted. The concentration in the kidney probably represents the end result of this process. Considering the above facts, it seems that this individual was suffering from lead poisoning though apparently not severely poisoned. The exposure could not have been severe or more lead would have been found in the bone in spite of the acidosis.

CASE 17. A male, 48 years of age, was employed as an underground cable-splicer using a blow torch on solder and lead sleeving for 20 years. He died one week after quitting his job but had suffered the following symptoms for two years: palsy, wrist-drop on right, ashy-gray color, loss of appetite, general swelling, pains in chest, dyspnea, and engorged vessels. Postmortem findings: bronchopneumonia, lympho-sarcoma of the mediastinum. The last four symptoms named must have been due to the sarcoma but the others can be ascribed to lead. He died from bronchopneumonia caused by the sarcoma.

CASE 18. A male, age 46 years, was employed at a lead refinery for seven years, but the exact type of work was unknown. He left work one week before his demise. There was a lead-line on the gums. Symptoms: chills, and coughed up blood.

TABLE X

TWENTY-ONE CASES OF INDUSTRIAL CHRONIC LEAD POISONING IN ADULTS

CONCENTRATION (mgm. Pb per 100 gm.)

Case No.	Exposure	Symptoms of Lead Intoxication	Liver	Kidney	Bone (Sternum and Ribs)
10	21 years at white lead company	Definite	0.40		
11[a]	Six years as painter	Complicated by gastric ulcers	0.40	0.80	
12[b]	15 years pouring solder	Unrevealed or complicated by carcinoma	1.39	0.20	7.84
13[c]	Eight months winding silk	Definite	0.60	0.40	
14[d]	18 years with white lead and lead arsenate	Definite	0.88	0.38	6.60
15	18 months welding white lead mixers	Complicated by peptic ulcer	0.80	0.62	8.00
16	Seven years soldering and scrapping radiators	Complicated by fibrinous plastic peritonitis	0.24	2.00	0.50
17	20 years splicing cables	Definite	0.74	0.42	1.21
18	Seven years at lead refinery	Unrevealed but indicated	2.55	0.79	
19	10 years as painter and decorator	Definite	0.93	0.69	3.40
20	Four years at smelting and refining company	Definite	0.80		2.81
21	18 months grinding and polishing soldered auto body seams	Possibly complicated by appendicitis	0.73	0.60	2.41
22	Seven years in battery plant	Definite	0.65	0.57	5.24
23	Four years at electrical manufacturing company	Somewhat doubtful	0.71	0.57	
24[e]	Six and a half years at paint factory	Definite	0.61	0.20	6.00
25	Seven years at red lead plant	Definite	1.80	0.40	9.20
26	Three months in casket factory	Complicated by appendicitis	0.60	1.08	
27	13 years at lead company	Complicated by adhesions and sarcoma	1.46		1.60
28	Six months at lead smelter	Definite	2.67	0.28	1.59
29	15 years at white lead company	Complicated by multiple adhesions	0.92	0.34	20.56
30	Lead-paste mixer at white lead company	Definite	0.64	0.11	5.60
	Mean		0.97	0.58	5.47
	Probable error of mean*		± 0.14	± 0.10	± 1.29

a—Lead in stomach, 0.40 mgm. per 100 Gm.
b—Lead in lungs, 0.24 mgm. per 100 Gm.
c—Lead in intestines, 0.40 mgm. per 100 Gm.
d—Lead in brain, 0.20 mgm. per 100 Gm.
e—Lead in brain, 0.40 mgm. per 100 Gm.
*No cases were omitted in this calculation.

Postmortem findings: lobar and bronchopneumonia, empyema of left pleural cavity, hypertrophy of left ventricle, infectious hyperplasia of spleen, catarrhal cystitis. He died of lobar and bronchopneumonia. The concentrations of lead in the viscera indicate a marked absorption of lead, and also a possible severe intoxication even though more complete information about the symptoms was lacking.

Case 19. A male, 31 years of age, was a painter and interior decorator for 10 years, using brush, rubbing down painted surfaces, and using spray-gun during his last year of employment. He had abdominal pains, vomiting spells, and headache for four years, shortness of breath four months, general weakness, swelling of scrotum, feet and arms two months, and many stippled cells. He quit work one year prior to death and was treated in a hospital during several months for plumbism. Postmortem findings: chronic nephritis, cardiac decompensation, hypertension. The cause of death was chronic nephritis, possibly caused by lead.

Case 20. A male, 30 years of age, was employed for four years at a smelting and refining company as a scale man, weighing and charging scrap lead and slag into a furnace. He became ill two days before death with abdominal cramps and general weakness, was treated for lead colic and sent home. He died suddenly. Postmortem findings: massive hyptertrophy of the heart. The immediate cause of death was hypertensive heart disease.

Case 21. A male, 30 years of age, was employed 18 months at an automobile assembly plant grinding down and polishing soldered seams in automobile bodies. Two months before death he left work complaining of severe abdominal pains and a swelling of the right leg. Many stippled cells were found by the plant physician. An appendectomy was recommended and performed. Death followed several days later. The autopsy revealed that the cause of death was a generalized peritonitis secondary to an opening in the caecum which developed after the appendiceal stump had ruptured. There was some doubt that an appendectomy had been necessary.

Case 22. A male, 40 years of age, was employed in a battery plant seven years, but his exact duties were unknown. He had two acute attacks of lead poisoning during his last year of life. Symptoms during the last three months away from the job: abdominal cramps, pain in extremities, swelling of ankles, irrational. There was a lead line and many stippled cells. Postmortem findings: bilateral suppurative pyelonephritis, bronchopneumonia, encephalopathy. The cause of death was uremia.

Case 23. A male, 37 years of age, worked four years at an electrical manufacturing company doing general labor. The history of exposure to lead was rather vague or at least unrevealed. Severe pain developed in the epigastrium two days prior to death. He entered a hospital and died one hour later. Postmortem findings: luetic aortitis, chronic myocarditis, edema of the lungs. The cause of death was luetic aortitis. It is a matter of conjecture as to whether or not the pain in the epigastrium was referred from the heart or was an acute attack of lead colic. The abnormal concentrations of lead found in the organs indicate lead intoxication, in the presence of which it is reasonable to believe that the pathologic condition was aggravated, but the same would apply to alcohol and other toxic agents, and over-exertion.

Case 24. A male, 37 years of age, handled red lead and white lead in a paint factory for six and a half years. He was seized with a severe attack of abdominal cramps, vomiting, and constipation 40 days before death and soon became very irrational. The records indicate an attempt to blame syphilis for his condition, but a negative Wasserman-Kahn and subsequent anatomical findings ruled this out. He died in an insane asylum. The postmortem findings revealed hemorrhagic encephalitis as the cause of death. The exposure, symptoms, and the analysis of the organs indicate that the encephalitis was due to lead.

Case 25. A male, 48 years of age, was employed at a red lead plant seven years, charging a litharge furnace. He had several attacks of lead colic within eight months and became incapacitated three months prior to death. Symptoms: abdominal cramps and constipation. There was a lead line on the gums. The autopsy revealed a bacterial (vegetative) endocarditis which was the immediate cause of death, but the symptoms and concentration of lead in the viscera indicate that he was severely poisoned with lead.

Case 26. A male, 25 years of age, worked in a casket factory for three months grinding down and polishing lead caskets and soldered seams of other metallic caskets. About 10 days before death severe abdominal cramps, vomiting and obstipation set in. Many stippled cells were found. He died following an appendectomy. Autopsy revealed an acute fibrinous peritonitis following the appendectomy as the immediate cause of death. The symptoms were probably due both to the appendix and the lead, but the chemical findings indicate that he was suffering from lead intoxication at the same time.

Case 27. A male, 43 years of age, worked at a lead company for 13 years. A piece of hot lead lodged in the inner corner of one eye; later, breathing became difficult, he had severe headaches, and lost his sense of smell. A sinus operation was performed and he was dismissed 1 week later in an improved condition. He returned to his old job at the lead plant and the following signs and symptoms developed within a few months: lead line, stippling, loss of appetite and again the sense of smell, severe headaches and general pains, and constipation. He died 10 months after the sinus operation. Postmortem findings: sarcoma of the ethmoid and turbinate, multiple sarcoma, and adhesions of the internal organs. Death was due to multiple sarcoma. It was said that sarcoma of the ethmoid and turbinate was caused by the piece of hot lead, which, if true, would appear to be due more to the irritating effect of the hot particle than to the toxic effect of absorbed lead.

Case 28. A male, 37 years of age, worked at a lead smelter for six months, tapping out metallic

lead from slag and molds, weighing the lead, and charging a "sweat-furnace." He had a severe attack of lead colic two months previous to death, was sent home and medical treatment administered. He returned to work and five days prior to death developed the colic described as follows: cramps in the abdomen, legs and arms occurring intermittently every five or six minutes. There was a definite lead line on the margin of the gums, but no stippled cells were found. Following this a cough, chills, high fever, and sweats developed. The autopsy showed the immediate cause of death to be lobar pneumonia, and there was also present a diffuse suppurative bronchitis. The lead in the bone is quite low but might be anticipated in an exposure of only six months duration. Deposition of lead in the liver is very marked, indicating a severe exposure at a recent date, and while not high in the kidney, is abnormal.

Case 29. A male, 54 years of age, worked at a white lead company 15 years. He handled white lead in the plant for 12 years, developed lead colic and was transferred to a job as janitor in the wash-room and locker-room, where he worked for three years longer. His last job was shaking and washing overalls of white lead workers, sweeping and mopping the locker-room. He worked until the day before he died, but frequently had abdominal pains and was constipated continuously. On Thanksgiving Day he drank a few beers and returned home at 2:00 in the afternoon complaining of severe abdominal cramps, weakness in the left arm, and was nervous and excited. To his family "he did not appear to be drunk." He was put to bed and found dead six hours later. The autopsy revealed the following: chronic myocarditis, hypertrophy and dilation of right and left ventricles, moderate sclerosis of coronary vessels, bilateral parenchymatous nephritis, bilateral obliterative fibrous pleuritis, fibrous and calcified perisplenitis, adhensions of diaphragm, colon and stomach to liver, adhesions of caecum and ascending colon to peritoneum, ancient removal of appendix. In the opinion of the physician who performed the autopsy, the adhesions were post-operative, and the fibrosis and calcification were due to some old infection such as typhoid or malaria. Ethyl alcohol by weight was determined in the organs with the following results: stomach and contents 0.09%, liver 0.08%, kidney 0.10%, indicating a very mild intoxication or only a slight incoordination at the time of death. However, within a period of six hours the percentage of alcohol in the viscera would decrease considerably and the chances are that he was in the excitable stage of alcoholic intoxication upon his arrival at home. The immediate cause of death was chronic myocarditis. The extensive adhesions and fibrosis in the internal organs were undoubtedly the cause of most of his complaints which were evidently accentuated by abnormal amounts of lead. He probably suffered an acute attack of lead poisoning precipitated by alcoholic intoxication just prior to death.

Case 30. A male, 36 years of age, was employed as a lead paste mixer at a battery company for an unknown period of time. Four months prior to death he suffered an acute attack of lead colic and hypertension, was treated in a hospital for one month and discharged. One month later he returned to the hospital with a similar attack, and died within two months. Postmortem findings: lobar pneumonia, chronic nephritis, chronic myocarditis. The cause of death was lobar pneumonia. The history of lead exposure, two attacks of lead colic, and abnormal concentrations of lead in the liver and bone indicate that his fatal illness developed during lead intoxication. The concentration of 0.11 mgm. of lead in the kidney is within the "normal" range, and is further evidence that an impaired renal function may result in a low concentration of lead in that organ.

Discussion: This group of 21 cases represents to a very large degree the number of justified cases of industrial plumbism that have occurred in Cook County, Illinois, over a period of ten years. The justified cases are approximatley one-third of the total number of suspected lead poisonings. The symptoms most frequently observed are abdominal cramps, constipation, vomiting, head-ache, vertigo, loss of appetite, general weakness, secondary anemia, and pallor; less frequently wrist-drop, palsy, encephalopathy, and metallic taste in mouth. Basophilic stippled cells may or may not be found, depending upon the stage and degree of intoxication. They are not a specific indication of lead poisoning because they are found in too many other conditions. The presence of an abnormal number of stippled cells is just another indication of intoxication, but if the count is normal or negative it must not be construed to mean that lead intoxication is absent.

The postmortem examination will reveal the cause of death, which in most instances has no direct relationship with lead poisoning insofar as we know at the present time because our knowledge of the pathology of the disease is very limited. Such lesions as encephalitis and nephritis can be ascribed to lead with a reasonable degree of medical certainty. The incidence of nephritis has been found to occur more frequently among lead workers than among non-lead workers.[61] However, we cannot prove beyond a reasonable doubt that any of the pathological conditions found at the autopsy were caused directly or indirectly by lead because we know such conditions exist when there is no lead intoxication. Proof beyond a reasonable doubt is unnecessary here because of the civil rather than criminal aspect of such cases. The autopsy may reveal that there were certain lesions or adhensions in the internal organs that were the cause of cramps, loss of appetite, vomiting and constipation. This might raise some doubt about the observed symptoms being caused by lead because the symptoms of lead intoxication were probably obscured by pathological conditions as found at the autospy. Both lead and disease could have been and probably were responsible for the observed symptoms. This is probably true in 50 per cent of the cases in this group and is a reason why the history of exposure and the first appearance of symptoms should be traced back to the very beginning.

The determination of lead in the organs by a qualified chemist or toxicologist is of the utmost importance, first, because it actually establishes the presence of lead, and, second, because it will reveal whether or not the concentration is of toxicological significance. The concentration of lead in this group has been shown to be associated with definite symptoms of plumbism almost invariably, and probably is the most reliable objective evidence of intoxication. Much of the other evidence may be hearsay. Symptoms may be feigned by the employee or overlooked by the plant physician for obvious reasons. Stippled cells may be found in abnormal numbers but are non-specific. What appears to be a lead line on the gums may be due to bismuth and it is a sign of absorption and not a symptom of intoxication. It is usually found in the margin of the gums at the base of decayed teeth or where there is poor oral hygiene which favors the formation of black lead sulfide. It is not seen where there is good oral hygiene or false teeth. It has been said that most people of the Negro race have a dark bluish-black line at the margin of the gums which cannot be distinguished from the Burtonian lead line. Some colored people have a generalized bluish tint to the entire gums, or there may be an outstanding purplish vein running parallel to the margin but higher up on the gum. If present, the lead line on the margin of the gums can be distinguished as easily in colored people as in whites. Most signs and symptoms are usually subjective. The postmortem findings are actually seen thousands of times when there is absolutely no evidence of lead intoxication. The pathologist may suspect that certain pathology was due to lead, but scientific proof is lacking without the aid of a chemist. It is only fair to consider the living victim of lead intoxication as an abnormal individual. He is sick because the normal equilibrium of his body and certain vital functions have been disturbed, thus lowering his resistance. While in this condition, he may succumb to some acute infection as pneumonia or influenza, or some pathologic condition which existed in a milder form before plumbism developed may be seriously aggravated by lead. Degeneration of tissue may proceed to a point beyond the ability of the body to readily repair or regenerate it as in cases of encephalitis or chronic nephritis wherein lead intoxication has been established.

We come to the conclusion that certain concentrations of lead have been observed to exert toxic effects in the human organism in a relatively large group of cases. Typical symptoms of intoxication have been found in a large majority. Therefore, the concentrations of lead in the respective tissues of this group are of toxicological significance. Further proof of this is the fact that no evidence of intoxication was found in 39 cases in which the concentration of lead was decidedly lower (14 suspected but unjustified, and 25 "normal" cases).

A conclusion of chronic lead poisoning is justified when the following reliable evidence is established.

1. A history of an abnormal exposure to lead.
2. Symptoms of lead intoxication.
3. Signs of lead absorption (not so important in fatal cases because the finding of lead in the tissues by chemical analysis is proof of absorption).
4. Anatomical diagnosis from postmortem findings.
5. Concentration of lead in the tissues, as summarized in Table X.

It is of prime importance to establish the fact that the victim was suffering from lead poisoning at the time of death, in which case lead becomes a material factor in the cause of death, unless death was due to external injury.

The statement "once leaded, always leaded,"[49] has its limitations. For example, the victims in Cases 10 and 19 had been removed from the exposure for periods of eight months and one year respectively, and were suffering from lead intoxication at the time of death, but we have no way of knowing what the symptoms or concentrations would have been had they survived a much longer period of time, say two or three years. It is also possible that some of the individuals in the unjustified cases (see Table XII) might have suffered a certain degree of intoxication at one time, but sufficient time had elapsed for the elimination of lead and disappearance of symptoms. The truth of the statement seems to depend upon the length and severity of exposure, and the health of the individual. It is true that lead will always be present in the tissues, but not in toxicologically significant amounts providing the individual has been removed from the exposure a sufficient length of time, which seems to be two or three years. Clinicians are said to have observed supposed recurrences of toxic episodes of plumbism precipitated by acute infections, or fractures 10 or 12 years after removal from a lead exposure. Such observations lack the necessary proof unless toxic quantities of lead have been found in certain body fluids or tissues at the same time.

There are no signs, symptoms, or anatomical findings pathognomonic of lead alone, and in many instances an abnormal exposure must be assumed. Therefore, the foregoing evidence must be correlated with lead concentrations in the tissues as determined by chemical analysis in order to diagnose the disease.

Five Doubtful Cases

IN THIS group are found four cases where the exposure was of an industrial nature and one case in which colloidal lead phosphate was administered as a therapeutic agent in the treatment of cancer. With the exception of Case 35 they are classified as doubtful for the following reasons:

1. The history of an abnormal exposure to lead was rather vague and very slight.
2. Symptoms of intoxication were not very characteristic.
3. The chemical findings, while somewhat above normal as compared to the values in Table XIII, were considerably lower than those in Table X where intoxication was established.

Here again we find that slight exposures and doubtful symptoms are correlated with significantly lower concentrations of lead in the tissues. It

TABLE XI

FIVE CASES OF DOUBTFUL CHRONIC LEAD POISONING IN ADULTS

CONCENTRATION (mgm. Pb per 100 gm.) in the tissues

Case No.	Exposure	Symptoms of Lead Intoxication	Liver	Kidney	Bone (Sternum and Ribs)
31	Janitor and painter	Not very characteristic	0.25	0.63	
32	Painting window shades	Not very characteristic	0.30	0.30	2.01
33	Machinery set-up man in can factory	Not very characteristic	0.39	0.39	1.34
34	Soldering tin cans	Not very characteristic	0.31	0.10	
35a	Injections of colloidal lead phosphate	Complicated	15.00*	2.52*	2.60*
	Mean		0.31	0.36	1.68
	Probable error of mean		$\pm$0.029	$\pm$0.11	$\pm$0.335

a—Lead in spleen, 15.00 mgm. per 100 gm. *—Omitted in calculation of the mean.

has been found that lead is always present, even in cases where there has been no abnormal exposure and absolutely no indication of intoxication, and that the lower the concentration of lead in the tissues, the less chance there is of intoxication. The results are summarized in Table XI.

CASE 31. A male, 45 years of age, had been a janitor most of the time, but because there was a history of some painting the organs were submitted for lead determinations. Symptoms: sudden seizure with a severe epigastric pain. Two stomach operations, pains and abdominal cramps from then on. The duration of illness was unknown. Postmortem findings: gastro-entero-anastomosis, chronic gastritis, erosion of serosa and subserosa, post-operative perforation into abdominal cavity, degeneration of internal organs, diffuse colloid goitre. The cause of death was gastric ulcers.

CASE 32. A male, 49 years of age, painted window shades with a brush. The duration of illness was unknown. Symptoms: spots before eyes, dizzy spells, palpitation, weakness, general edema, coughed blood. Postmortem findings: degenerative heart disease, hemorrhagic infarcts in lungs,

TABLE XII

THIRTEEN CASES OF SUSPECTED BUT UNJUSTIFIED CHRONIC LEAD POISONING IN ADULTS

CONCENTRATION (mgm. Pb per 100 gm.) in the tissues

Case No.	Exposure	Symptoms of Lead Intoxication	Liver	Kidney	Bone (Sternum and Ribs)
36	Washed pig lead in vats of water	Ruled out by other findings	0.30	0.20	
37	One day in steel plant, thought to be lead factory	None	0.40	0.10	0.20
38	Painted trucks with brush 18 years	Ruled out by other findings	0.15	0.10	2.14
39	Vague history of painting	Ruled out by other findings	0.16	0.12	0.64
40	Stenciled enamelware with powders three months	Ruled out by other findings	0.13	0.06	0.50
41	Teacher of printing-press work five years	None	0.24	0.18	0.75
42	Furnace tender seven years, unemployed five years	None	0.33	0.13	
43	Painter, number of years unknown	Ruled out by other findings	0.20	0.17	1.65
44	Handled automatic shellac dipper of metal pieces 10 years	Ruled out by other findings	0.15	0.08	0.46
45	Painter, number of years unknown	None	0.08	0.04	1.31
46	Dross skimmer, flue cleaner at lead lead plant two weeks	Ruled out by other findings	0.07	0.16	0.09
47	Body-assembly man in can factory 18 mos. Lit solder pots	Ruled out by other findings	0.09		0.86
48	Printer for many years	Ruled out by other findings	0.21	0.05	1.64
	Mean		0.19	0.11	0.93
	Probable error of mean		$\pm$0.025	$\pm$0.016	$\pm$0.199

chronic duodenal ulcer. The cause of death was degenerative heart disease.

Case 33. A male, 46 years of age, changed and set up machinery for making cans, for ten months. The only known exposure to lead was a pot of molten solder in the same room, but he did not work with the solder. The duration of illness was about two days and came on during an extreme heat wave. Symptoms: cramps in legs, arms and abdomen. Postmortem findings: hypertrophy of myocardium, fibro-plastic thickening of valve leaflets, atherosclerosis of aorta and coronaries. Cause of death: chronic myocarditis. The symptoms were probably precipitated by the extreme heat.

Case 34. A male, 33 years of age, soldered bottoms on tin cans for one year. He was ill two months. Symptoms: weak, irrational, headaches, disoriented, right side numb, pain in ear, ataxia. Postmortem findings: endocardial sclerosis, dilatation of heart, disseminated focal encephalomalacia. Cause of death: chronic endocarditis.

Case 35. A female, 40 years of age, was exposed to lead in the form of nine intravenous injections of 0.5% colloidal lead phosphate in 1000 cubic centimeters of 5% glucose over a period of 15 days. The calculated amount of elemental lead given was approximately 300 milligrams. She had a recurrent carcinoma of the breast, in treatment of which the lead preparation was administered, the rationale of this therapy being based upon the work of Bischoff and Blatherwick.[50] There may be some question why this case is placed in this group because it has been found that colloidal lead phosphate is relatively non-toxic. The reason is that agonizing abdominal cramps simulating acute lead colic were observed after the first, seventh, and ninth injections, and the cramps were relieved by the administration of calcium gluconate and morphine. The patient was in a state of cachexia and acidosis which perhaps increased the solubility and toxicity of the lead phosphate. Death occurred four weeks after the last injection. Postmortem findings: recurrent carcinoma of breast, generalized metastases. Cause of death: carcinoma of breast. Unfortunately the cancer tissue was not submitted for analysis. It would have been interesting to find out if lead showed any particular affinity for it, although Kehoe[22] found that it did not. Storage in the bone was not remarkable due to the time factor. The whole liver (780 grams) and spleen (175 grams) yielded 117 and 26.3 milligrams of lead respectively, approximately 48% of the administered lead. It appears that this remarkable retention of lead when administered as the colloidal phosphate is evidence that this is the form to which most of the lead is converted in the blood stream when absorbed, and is one of the mechanisms of protection that the human body has against lead poisoning. This patient showed a much greater retention of lead in the kidney than either of Kehoe's cases, one of which received colloidal lead phosphate with very little retention in the kidney, the other colloidal metallic lead with a much higher renal concentration.

In view of these facts, colloidal lead preparations should be administered with caution. The physician should be on the lookout for any symptoms of lead intoxication.

Discussion: Case 35 has been excluded in the calculation of the average concentrations for obvious reasons. The average concentration of lead in the liver, kidney, and bone is only 31, 50 and 30%, respectively, of the corresponding values in the preceding group of chronic cases. This fact, together with less hazardous exposures to lead and symptoms which were not very characteristic, seems sufficient to classify these as doubtful cases.

Fourteen Unjustified Cases

IT OFTEN becomes the duty of the toxicologist to rule out the possibility of lead poisoning when there is a history of a certain degree of exposure to lead, no matter how slight. For this reason the cases in this group are presented. They have the following characteristics: first, the exposure to lead was either very slight or practically negligible; second, the clinical findings and symptoms were closely associated with some demonstrable pathological condition, or external injury; and third, the concentration of lead, practically the same as the "normals" (see Table XIII), as found by a chemical analysis of the tissues was entirely too low to indicate lead intoxication.

Case 36. A male, 54 years of age, was employed in a lead refinery washing pig lead in a vat of water for an unknown period of time. He was incapacitated for one month. Symptoms: weakness, inability to walk, pains over iliac crests radiating to back. Postmortem findings: bronchopneumonia, osteomyelitis in lumbar area, chronic encephalomeningitis, cholelithiasis, late lues. Cause of death: bronchopneumonia.

Case 37. A male, 49 years of age, worked in a steel plant one day, originally thought to be a lead plant. Died suddenly. Postmortem findings: gumma of heart, aneurism of aorta, chronic adhesive pericarditis, proliferative peritonitis. Cause of death: gumma of heart.

Case 38. A male, 57 years of age, painted trucks with brush 18 years for express company. He was incapacitated for one month. Symptoms: pain in side, weakness in limbs and feet. Postmortem findings: melanosarcoma. Cause of death: melanosarcoma.

Case 39. A male, 47 years of age, painted at one time and was a known heroin addict for 20 years. He was one of about 16 who contracted malaria by passing around the "needle" among members of the "dope ring." He had gonorrhea and lues, scrotal hernia, swelling of the feet, was irrational and comatose. Postmortem findings: infectious (malarial) hyperplasia of spleen, subacute glomerulonephritis, inguinal hernia, bronchopneumonia. Cause of death: malaria. Tests for morphine derivatives and other alkaloids in the tissues were negative.

Case 40. A female, 18 years of age, stenciled letters on vitreous enamel ware with colored powders for three months. Some of the powders were found to contain small amounts of lead, apparently as contamination. She was ill four months. One month prior to death 0.07 mgm. of lead was found

TABLE XIII

A SUMMARY OF LEAD CONCENTRATIONS IN TISSUES OF ALL GROUPS

(74 cases)

Classification	No. of Cases	Range (Mgm. Pb/100 gm.)			Average Concentration (Mgm. Pb/100 gm.)			300-gm. composite liver, kidney, bone. Total Pb
		Liver	Kidney	Bone	Liver	Kidney	Bone	
Normal Fetal	5	0.011–0.049	0.014–0.028	0.005–0.110	0.036	0.021	0.052	0.109
Normal Childhood	5	0.050–0.297	0.038–0.143	0.524–3.112	0.18	0.09	1.54	1.61
Normal Youth	5	0.074–0.237	0.042–0.168	0.448–2.032	0.15	0.08	1.07	1.30
Normal Middle-Age	5	0.112–0.307	0.054–0.115	0.598–1.528	0.23	0.10	1.07	1.40
Normal Old-Age	5	0.038–0.208	0.029–0.108	0.398–2.025	0.10	0.06	1.35	1.51
Unjustified Chronic Industrial	13	0.07– 0.40	0.06 –0.20	0.09 –2.14	0.19	0.11	0.93	1.23
Doubtful Chronic	4	0.25 –0.29	0.30 –0.63	1.34 –2.01	0.31	0.86	1.68	2.35
Chronic Industrial	21	0.24 –2.67	0.11 –2.00	0.50 –20.56	0.97	0.58	5.47	7.02
Chronic Non-Industrial in Children	9	0.74 –6.60	0.16 – 3.10	0.85 –13.47	3.13	1.43	5.38	9.94
Acute	1	9.70	1.15	3.56				14.41
Subacute Therapeutic	1	15.00	2.52	2.60				20.12

Observe the relationship between the total lead content of the composite specimens in the last column, and the presence or absence (as the case may be) of lead intoxication.
The lead content of the unjustified group is within the normal limits.
The lead content of the doubtful group is about twice the normal.
The lead content of the chronic group is about five times the normal.
In children and in the acute case it is significantly higher than in the industrial groups.

in a 24-hour specimen of urine, which amount is within the normal limits. There was an enormous edema of the trunk and extremities. Postmortem findings: glomerulo-nephritis, fibrous adhensions between heart and pericardial sac. Cause of death: extensive chronic glomerulo-nephritis.

CASE 41. A male, 63 years of age, taught the art of printing press work five years in public schools. One week before death he tripped over a stool at the school, fractured several ribs, and developed severe headaches and a pain in his side. Postmortem findings: fractured ribs, cardiac failure, arteriosclerosis. Cause of death: arteriosclerotic brain disease and cardiac failure complicated by fractured ribs.

CASE 42. A male, 50 years of age, was a furnace-tender for seven years, handling coal and ashes. He was out of work five years, and bed-ridden one year with symptoms of tuberculosis and rheumatism. Postmortem findings: hypertrophy of myocardium, rheumatoid deformitus in joints, anthracosis, anthraco-tuberculosis. Cause of death: chronic myocarditis and coronary sclerosis. The lungs were black with coal dust but the determination of carbon was not performed. The silica content of the lungs was quite high, 1 gm. of dry lung tissue yielded 6.41 mgm. SiO_2, and the lung ash yielded 8.06% SiO_2. Apparently the high silica and carbon content predisposed to tuberculosis.

CASE 43. A male, 40 years of age, was a painter for years. He was picked up on the street in a coma and lived two days. He had several convulsions. There was a flaccid paralysis of the left side and ptosis of right eye lid. Postmortem findings: skull fracture and cerebral lacerations as the cause of death.

CASE 44. A male, 46 years of age, was first reported as having been a painter for 10 years. Investigation revealed that his job was to hang metallic pieces on an automatic conveyor which dipped the pieces into vats of varnish and shellac. He was ill for two weeks. Symptoms: paralysis of

TABLE XIV

FIVE CASES OF NON-FATAL LEAD ENCEPHALOPATHY IN CHILDREN

Case No.	Lead Exposure	Time	Signs and Symptoms[a]	Mgm. Pb/10 cc. Blood
1	Two year old boy. Mother burned storage battery cases	6 mos.	1, 3, 5, 16, 17, 19	0.19
2	Five mos. old boy. Mother used lead nipple shield	2 mos.	1, 3, 16, 17, 19	0.24*
3	One year old girl. Mother burned storage battery cases	6 mos.	5, 6, 16, 17, 19	0.32
4	Two year old girl. Same exposure as No. 3	6 mos.	1, 3, 16, 17, 19	0.08
5	One and a half year old boy. Gnawed crib posts (pica)		16, 17	0.06

*—Many basophilic stippled erythrocytes. a—See reference table of numerals under Table XV.

upper and lower extremities, secondary anemia, diarrhea. Postmortem findings: hypertrophy of myocardium, bronchopneumonia, hyperemia and edema of brain and lungs. Cause of death: bronchopneumonia complicating chronic myocarditis.

Case 45. A male, 60 years of age, had been a painter for years and died suddenly. Postmortem findings: coronary occlusions and atherosclerosis. Cause of death: coronary occlusions.

Case 46. A male, 27 years of age, was a dross-skimmer at a lead plant for two and one-half weeks, and cleaned flues. During the third week at work, cramps and vomiting set in. A 24-hour urine specimen taken while on the job showed 0.10 mgm. of lead per 1000 cc. and a very large amount of bile pigment. No stippled red cells were seen. During two weeks in a hospital he became extremely jaundiced, and could not digest fats which when eaten caused vomiting and constipation. He became irrational and died. Postmortem findings: red atrophy and focal degeneration of liver, hemorrhagic bronchopneumonia, subendocardial hemorrhages, edema of brain. Cause of death: acute red atrophy of the liver.

There was a history of syphilis three years prior to this but he had been pronounced "cured" and had received no arsenic, mercury, or bismuth within a year. In the opinion of the late Dr. R. H. Jaffé, Chief Pathologist of the Cook County Hospital, the red atrophy apparently started just previous to or at the time this man began work. The cause of this condition was unknown but evidently was not due to lead. For some unknown reason the concentration of lead in the bone was the lowest of all adults examined.

Case 47. A male, 29 years of age, was a body-assembly man in a can company for 18 months, and also started fire under solder pots in the mornings. He became ill with extreme vomiting, diarrhea, and a marked secondary anemia. No stippled cells were seen. Three days before death a 24-hour specimen of urine showed 0.007 mgm. of lead per 1000 cc., exceptionally low, perhaps due to impaired renal function. Postmortem findings: congenital atrophy and malformation of both kidneys and ureters. Cause of death: uremia.

Case 48. A male, 60 years of age, had worked as a printer for 40 years. He complained of pains in the abdomen and right leg, vomiting, had diarrhea for two days, and died four days later. He had a similar attack six months prior to this. Postmortem findings: bilateral focal and confluent bronchopneumonia, fat necrosis of the pancreas, perforated ulcer of the stomach with phlegmonous gastritis. Cause of death: bronchopneumonia.

Discussion: The evidence speaks for itself—that a conclusion of chronic lead poisoning in any of these 13 cases would be unjustified.

The concentrations are of the same order as the corresponding values obtained in "normal" individuals. If the chemical findings only were available in a case similar to these, a toxicologist could predict with a reasonable degree of certainty that the chances of lead intoxication were extremely slim. Again we find a close correlation of the chemical findings with the other evidence.

An interesting case is that of J. B., a white male, 35 years of age, whose death occurred too late to insert the case in its proper place here. The history revealed that he had painted and lacquered automobile winter fronts, slot-machines, and novelties for several years. Due to this fact his was a suspected case of plumbism. He complained of general weakness, especially in the arms and legs, which condition grew worse and developed into a progressive muscular atrophy. On 12-18-37 a 24-hour specimen of urine yielded 0.067 mgms. Pb per 1000 cc., which amount is within the normal limits. On 3-1-38 a 24-hour specimen of urine yielded 0.02 mgm. Pb per liter, after receiving 60-80 gr. NH_4Cl per day for three days. From these results the writer offered the opinion that the patient was not a victim of plumblism at this time, which opinion was not concurred in by the attending neurologist. Death occurred on 9-26-38, the cause of which was bronchopneumonia complicated by chronic myocarditis. The liver yielded 0.19, the kidney 0.06, and the bone 0.84 mgm. Pb per 100 grams, well within the normal limits, and ruling out the possibility of plumbism. Again we find that the concentration of lead in the urine is a reliable indicator of that in the tissues.

A Case of Acute Lead Poisoning

Case 49. A woman, 49 years of age, died two days after a gall-bladder operation. Two days prior to the operation she was said to have partaken of a six-ounce bottle of medicine, four ounces of which were missing from the bottle. The medicine was found to be a mixtures of iodides, bromides, and salicylate, and contained 1.5% lead calculated as metallic lead. There was a yellowish-brown precipitate in the mixture, apparently lead iodide, but the lead was probably originally present as lead acetate.

Five months after burial the embalmed body was exhumed and an autopsy performed. The condition of the tissues was said to be such that the immediate cause of death could not be determined from the anatomical findings. The following concentrations of lead (mgm. per 100 gm.) were found by chemical analysis: liver, 9.70; kidney, 1.15; bowel and contents 18.48; bone (femur), 3.56. A large amount of bismuth was found in the bowel, but traces only in the liver and kidney.

The large amount of lead in the bowel shows a recent swallowing of a toxic quantity. This fact, together with a significantly lower concentration in the femur, is indicative of an acute poisoning. Lead in the liver was 10 times higher and in the kidney twice as high as the respective concentrations in the chronic industrial group (Table X). There can be no doubt that this woman was suffering from the effects of acute lead poisoning at the time of death.

Forty-Six Cases of Non-Fatal Lead Poisoning

Five Cases of Lead Encephalopathy in Children

These are well-defined cases of lead poisoning, characterized by the rapid onset of acute symptoms after relatively short periods of expo-

sure. The acute episodes of poisoning are more severe than in the adult cases, but this does not classify them as acute poisoning because absorption occurred over a period of several weeks to several months. The clinical mainfestations were malaise, anorexia, loss of weight, colic, headache, and constipation. They became nervous and irritable. Some had projectile vomiting. Drowsiness and stupor developed in association with generalized clonic convulsions. One child had a convulsion for 15 minutes after admittance to the hospital. Strabismus, nystagmus, and a pill-rolling motion of the fingers were frequently observed.

In each case abnormal amounts of lead were found in the blood and the x-ray revealed lines of increased density at the ends of the long bones. The roentgenologic diagnosis of lead poisoning in children has been advocated by Park,[51] Vogt,[52] and many others. Bucy and Buchanan[53] reported three cases of lead encephalopathy with increased intracranial pressure simulating intracranial tumors, and a marked spread of the sutures of the skull was observed. In two of their cases suboccipital decompressions were made with prompt and complete recovery. The third was put on medical management alone, but "this child had a prolonged and stormy course but also ultimately recovered." They believe that medical management is "notoriously unsatisfactory and results in a high mortality and numerous and serious sequelae." In three of the nine fatal cases reported in the present paper, subtemporal decompressions were made but the results were obviously unsatisfactory. The five non-fatal cases reported here were all on medical management.

Recovery was slow but it cannot be foretold at this time just how numerous and serious the sequelae will be.

Recently Blackman[54] made a clinical and pathological study of 22 cases of fatal encephalitis in children. He found that the brain was swollen and may be hyperaemic, or pale. Numerous lesions were found throughout the entire central nervous system, mostly in the cerebral hemispheres and cerebellum, and "the microscopical changes were those of a serous inflammation. Most of the injury to the nervous tissue seems dependent on the accumulation of exudate."

The serious consequences and frequency of this disease in children show the necessity for eliminating lead in any form from the home. The owners of storage battery plants and smelting and refining works should be required to make their piles of old battery cases inaccessible to any persons desiring to use them as fuel. Municipalities should prohibit the manufacture and sale of lead toys, casting sets, and all children's furniture finished with a lead-containing paint.

The results of the work on blood indicate that any figure above 0.05 mgm. per 100 cc. is indicative of lead intoxication in children. Those reported in Table XIV are the first specimens drawn after admission to the hospital. During the course of illness other analyses were made, and the result in most instances was 0.10 mgm. or more while suffering from the disease.

Children seem to be particularly susceptible to the toxic effects of lead, perhaps due to the tenderness and low resistance of the young body cells. They appear to be more acutely ill than adults. However, we must consider the fact that because they are totally unaware of the hazard, more lead is absorbed, and much more recovered from their tissues than from adults.

Forty-one Cases, Non-Fatal Industrial, Chronic

THE first 31 men of this group were employed in a smelting and refining works, where the great bulk of the metal handled was lead. The work consisted mainly of the recovery of lead from battery plates, other scrap metal, and slag, in the manufacture of pig lead. An effort had been made to reduce the hazard, but the construction of the plant made this exceedingly difficult.

A plant physician had been on the job for 12 years, making and examining blood smears on all employees once a month, and making a physical examination of those who appeared to require it once a week. The fact that 31 of the 200 employees, representing 15.5%, were victims of lead poisoning during the period of one year is concrete evidence that the health of these men was not adequately protected. The time off from work varied from one and a half to two months. All of the victims received compensation during illness, and medical treatment. A few were discharged, some resigned, and others are still employed. In most instances a check-up was made, after time off and treatment by the physician, and it was found that the reticulocyte count had decreased by one-half, or had become normal. In the opinion of the plant physician,* a count of 6,000 per cu. mm. or below is as near normal as can be had in lead workers with no symptoms, but above 7,000 they begin complaining.

The "Basophilic Aggregation Test" of McCord[55] was used in examining the blood. This test represents a count of the total reticulocytes (young erythrocytes) in which is also included, but not differentiated, any stippled cells (punctated basophilic erythrocytes) that might be present. It is questionable whether the disappearance of symptoms and lowered reticulocyte count after treatment and time off was due to the medication, or removal from the lead exposure.

The "de-leading" treatment prescribed by the physician for men still on the job and not complaining was ammonium chloride tablets (5 gr. TID), or five drops of saturated potassium iodide solution in milk plus ¼ teaspoonful of sodium bicarbonate (TID). Saline cathartics such as magnesium sulfate and sodium sulfate were also used. The "demobilizing" treatment for men with colic was sodium thiosulfate (7½ gr.), or calcium gluconate intravenously.

Lead determinations in the urine were made by the writer because the plant physician had observed manifestations of lead intoxication, and because the men were complaining of illness.

The other 10 men (cases 32 to 41) were either

* Dr. F. J. Norton.

sent to, or voluntarily came to, the Cook County Hospital. In each instance clinical manifestations of lead poisoning were observed, and an abnormal lead exposure was verified.

It is unfortunate that the "stippled" cell counts were not made more often. In hospitals it seems that the physicians and technicians are interested principally in finding "stippled" cells by means of Wright's stain. Plant physicians usually use McCord's test for the total reticulocyte count, and in many instances report their findings in terms of "stippled" cells. This leads to unnecessary confusion and shows the need of a chosen standard to which all should adhere.

The concentration of lead in the urine was found to have an average value of 0.28 mgm. per 1,000 cc., which is approximately nine times the average "normal" value (Table VIII). The lead range was 0.08 to 0.76 mgm., but in 80% of the cases it was 0.15 mgm. or more.

As a general rule, when the concentration of lead in urine exceeds 0.20 mgm., the individual is quite severely poisoned with lead as verified by clinical evidence. If this value varies from about 0.08 to 0.20 mgm., it then becomes more difficult for the toxicologist to predict lead intoxication. However, if a somewhat low concentration is correlated with a history of abnormal exposure and reliable clinical evidence, a diagnosis of lead intoxication is established.

The Diagnosis of Lead Poisoning

THE mere fact that an individual is complaining of illness and has been working in a lead plant is not proof that he is suffering from plumbism. If aware of the hazard it may be a psychological effect, another illness, or he may be feigning illness. Symptoms may be present but they may be due to pathological conditions in which lead played no part. The basophilic aggregation test may be high, but this too may be due to other causes. The same is true of the stippled cell count. The mere demonstration of the presence of lead in the urine or blood is not proof of an abnormal absorption or intoxication because lead is known to be present normally. What, then, is the best way to diagnose a case of lead poisoning? Is there any standard to go by? The answer is that each case must be studied individually and all findings correlated.

Space does not permit a discussion of all signs and symptoms, but the industrial physician or medicolegal expert will readily recognize the fact that none of them are pathognomonic of lead alone. They may be present in other diseases or caused by poisons other than lead.

Colic (abdominal cramps) is generally conceded to be the symptom most frequently associated with lead poisoning. Lead causes a contraction of the gut (smooth muscle), which results in severe cramps, and constipation. This is found invariably among victims of plumbism, but is also caused by other diseases and poisons.

Loss of muscular strength and wrist-drop may or may not be due to lead. A deficiency of vitamin B in the diet can lead to a similar condition. Similar findings have been observed in chronic cases of poisoning due to arsenic, and tri ortho cresyl phosphate.

Tremor, palsy, and twitching of the muscles of the face may be seen, but chronic alcoholism must be ruled out. A similar case was seen recently in the hospital. He gave a history of painting occasionally, but the chemical findings did not indicate lead poisoning. A subsequent investigation revealed that the patient was a chronic alcoholic. Neurological symptoms should be thoroughly investigated as to the history of syphilis, alcohol, drug addiction, diet, habits, external injury, and hereditary influence.

Plant physicians have reported that negative Wassermans are frequently obtained on known syphilitics after they have worked for a while in a lead plant. This should not be construed to mean that lead may be a cure for syphilis, in spite of the fact that other heavy metals such as arsenic, mercury, and bismuth are used for this purpose. Some European workers, as well as Americans, have found that lead does not produce a negative result, and others believe its presence will yield a positive Wasserman.

The following signs and tests will be treated in greater detail because the apparent confusion about them seems to warrant explanation.

1. The Basophilic Aggregation Test (McCord's).[55] The blood smear is made in the usual way but the red blood cells are hemolysed with distilled water or hypotonic salt solution, and then stained with brilliant cresyl blue or Manson's methylene blue. The dye may be incorporated in the hypotonic solution so that hemolysis and staining is completed at the same time. This procedure causes the basophilic material within the cell to clump together or aggregate in vitro so that it can be more easily seen, hence the term "basophilic aggregation." McCord considered one such cell in every five fields as normal, and four to six in every five fields among lead workers as indicative of plumbism.

The apparent reason why so much confusion has arisen over stippling is because the test reveals the basophilic material in punctated or stippled form. The term "stippling" should not be used here because it is produced in vitro by artificial means. It includes the artificially formed stippled cells, as well as stippled cells which were preformed in the blood in vivo, but by this method the two cannot be differentiated. Therefore the count should be reported as "total reticulocytes" or "total immature erythrocytes."

It is true that the reticulocyte count increases among individuals suffering from lead poisoning, and it is a rapid laboratory test. It should not be used as a criterion of lead absorption or intoxication because the same picture is seen in secondary anemias, primary anemias, malaria, and metastatic carcinomas; in chronic poisonings from arsenic, or aniline derivatives. The test shows the response of the bone marrow in regenerating and

TABLE XV

FORTY-ONE CASES OF NON-FATAL CHRONIC LEAD POISONING IN ADULTS

Case No.	Place of Employment	Duties	Time	Signs and Symptoms[a]	Total reticulocytes (McCord's test) Per Cu. Mm.	Per Cent	Stippled Reticulocytes	Mgm. Pb/ 1000 cc. Urine
1	Smelting and refining works	Crane man	2 yrs.	1, 2, 3	10,000	2.0		0.12
2	Smelting and refining works	Unloading battery plates	3 yrs.	2, 5, 7, 8, 11	9,800	2.0		0.29
3	Smelting and refining works	Furnace man and janitor	7 yrs.	3, 14	9,000	1.8		0.10
4	Smelting and refining works	Blast furnace man	3 yrs.	1, 2, 3, 15	12,000	2.4		0.27
5	Smelting and refining works	General labor	8 mos.	1, 2, 7	8,000	1.6		0.17
6	Smelting and refining works	Charging blast furnace	6 mos.	1, 2, 4, 8	7,000	1.4		0.53
7	Smelting and refining works			3	6,500	1.3		0.53
8	Smelting and refining works	Furnace man	9 yrs.	1, 2, 3, 15	11,000	2.2		0.18
9	Smelting and refining works	General labor	6 mos.	2, 3	8,200	1.6		0.19
10	Smelting and refining works	Crane man and blast furnace	8 mos.	1, 2	8,000	1.6		0.23
11	Smelting and refining works	General utility	6 yrs.	1, 2, 3, 8	9,000	1.8		0.72
12	Smelting and refining works	General labor	3 mos.	1, 2, 4	6,200	1.2		0.14
13	Smelting and refining works	Furnace tapper	7 yrs.	1, 2, 3, 15	12,000	2.4		0.38
14	Smelting and refining works	General labor	5 mos.	1, 14	7,000	1.4		0.15
15	Smelting and refining works	General labor	4 mos.	1, 3, 7	6,500	1.3		0.16
16	Smelting and refining works	General labor	2½ yrs.	1, 2, 3, 7, 8, 11, 12, 15	13,000	2.6		0.12
17	Smelting and refining works	Furnace tapper	8 yrs.	1, 2, 7, 8	9,000	1.8		0.21
18	Smelting and refining works	General labor	2½ yrs.	3	6,500	1.3		0.43
19	Smelting and refining works	Foreman	7½ yrs.	7	5,500	1.1		0.45
20	Smelting and refining works	Blacksmith and cupel man	7½ yrs.	1, 2, 3, 7, 15	12,000	2.4		0.21
21	Smelting and refining works	General labor		1, 3	5,200	1.0		0.16
22	Smelting and refining works	General labor	3 mos.	1, 3, 7	5,000	1.0		0.22
23	Smelting and refining works	Furnace tapper	12 yrs.	1, 3, 7, 11	7,800	1.6		0.08
24	Smelting and refining works	General labor	4 mos.	1, 7, 11, 12	6,100	1.2		0.10
25	Smelting and refining works	Unloading battery plates	4 mos.	3, 7	6,200	1.2		0.59
26	Smelting and refining works	General labor	1 mo.	3, 7, 8	5,000	1.0		0.37
27	Smelting and refining works	General labor	4 mos.	2, 3, 7	6,300	1.3		0.12
28	Smelting and refining works	General labor	3 mos.	3, 7, 8	10,000	2.0		0.15
29	Smelting and refining works			1, 4, 7, 8, 12	8,000	1.6		0.21
30	Smelting and refining works			1, 2, 3, 7, 8	8,000	1.6		0.15
31	Smelting and refining works			1, 3	7,200	1.4		0.08
32	Smelting works	Cupola tender		1, 10, 15, 19			300	0.25 (0.19 mgm/100 cc. blood)
33	Smelting works	Charging furnace		1, 10, 18 3 attacks			Few 1st, none 2nd, 3rd	0.24
34		Painter	2 yrs.	1, 3, 5—2 attacks			200	0.20
35		Metal finisher		1, 4, 5			None	0.39
36		White lead painter		1, 12			33	0.33
37	White lead dept. paint factory	Tap-out man		1, 3, 5, 6, 15			A few	0.27
38	Solder foundry	Pouring	6 mos.	1, 3, 5, 7, 8, 9, 19			A few	0.33
39	Smelting works	Trucking	4 yrs.	1, 3, 13, 15			A few	0.76
40	White lead company	Barelling	8 mos.	1, 3, 5, 9, 13, 15			Many	0.31
41	Sign company	Lead chromate painter	20 yrs.	1, 2, 3, 5, 12, 19			Many	0.47
	Mean							0.28
	Probable error of mean							±0.027

[a] — Explanatory table of numerals:

1. Colic (abdominal cramps)
2. Abdominal tenderness
3. Constipation
4. Diarrhea
5. Nausea
6. Loss of appetite
7. Headache
8. Vertigo
9. Loss of muscular strength
10. Wrist drop
11. Tremor
12. Anemia
13. Pallor
14. Dyspepsia
15. Blue line on gums
16. Lead line in epiphyses of long bones
17. Encephalopathy
18. Neuritis
19. Excitability

pushing juvenile red blood cells into the blood stream to take the place of those destroyed in the capillaries. Therefore, it should be used as a measure of blood regeneration resulting from peripheral blood destruction which may be caused by many agents, among which is lead.

2. Basophilic Stippled Cells. When a blood smear is stained by Wright's method all erythrocytes are stained, including the reticulocytes. If there are any preformed stippled reticulocytes present, they will show up as such, but the fibrils of the other reticulocytes will be invisible. Stippled cells found in this way are said to be preformed in the blood, because they cannot be produced by adding lead in vitro. Because the basophilic material within the cells has been stippled or punctated in vivo, they should be reported as "stippled cells" entirely differentiated from the "basophilic aggregation."

The objection is often raised that they are too hard to see with Wright's stain. This is true if the smear is too thick. They are easily seen on a properly stained thin smear where there is only one layer of cells in the same plane.

Stippled cells are often seen in cases of lead poisoning, and are thought to be caused by the coagulating effect of lead in vivo on the intracellular basophilic material of certain reticulocytes.

They cannot be considered as diagnostic of lead poisoning any more so than the basophilic aggregation test because they are not specific for lead. The same diseases and poisons which cause one may also produce the other. They are usually found in the later stages of intoxication, but may not be seen at all. The peripheral blood is so transitory that unless the smear is made at the proper time (which is unknown) no stippling may be seen. A case may show two or three stippled cells per high-power field today and none tomorrow.

Their chief diagnostic value lies in the fact that if they are seen, toxic amounts of lead may be present, but a negative results does not rule out lead intoxication.

3. Blue Line on the Margin of the Gums (Burtonian Line). This is apparently a precipitation of bluish-black lead sulfide, by the reaction of hydrogen sulfide from decomposed sulfur-containing foods, on lead in the thin-walled capillaries and mucous membranes. It is found in about one case out of five, and only where there is poor oral hygiene; never seen with false teeth or good oral hygiene. It is not specific for lead because bismuth will produce a similar line.

The blue line is a sign of lead absorption, and not intoxication.

4. Line of Increased Density at the Epiphyses of Long Bones (by x-ray). This is found only in the bones of growing children, never in the adult. This area of increased opacity to x-rays was found by Aub, Robb, and Rossmeisl[24] to contain five times as much lead as the rest of the bone. They found the deposition of lead to go hand in hand with the deposition of calcium in this area of growth. The lead line is sharper and lacks the fuzziness of that seen in rickets. It is an important diagnostic feature of lead poisoning in children.

5. The Concentration of Lead in Urine. The results of the present investigation demonstrate that the concentration of lead in urine is the safest indication of intoxication.

It has been shown in Table VIII that the "normal" value for urine is approximately 0.03 mgm. per 1,000 c.c., ranging from 0.007 to 0.072 in persons with no symptoms of intoxication. This value is evidently closely correlated with an average of about 0.2 in the liver, 0.1 in the kidney, and 1.0 mgm. in the bone as indicated for normal adults in Table XIII. Lead within this range is apparently of no toxicological significance. It seems that up to this point excretion keeps pace with absorption, thus avoiding further accumulation. If exceeded and allowed to proceed long enough, the body is no longer able to maintain the equilibrium, absorption exceeds elimination and results in the accumulation of lead and a cumulative effect.

When absorption exceeds excretion, intoxication sets in as manifested by one or more of several signs or symptoms. At this point the concentration of lead in the urine was found to vary from 0.08 to 0.76 mgm. About 20 per cent of the cases ranged from 0.08 to 0.14 mgm., but intoxication was established by clinical evidence. In 80% of the cases a value of 0.15 mgm. or more was found, and intoxication was definitely established. The average in 41 cases of definite lead poisoning was 0.28 mgm.

It does not necessarily follow that any case coming within this range is definitely one of plumbism. The factor of individual susceptibility must be considered. Some persons require a longer time to show symptoms with a high concentration than others do with less. However, it is evident that abnormal excretion corresponds to abnormal absorption, and any case so characterized is an incipient or potential case of plumbism, if allowed to remain in the abnormal exposure.

The determination of lead in the urine is of the utmost importance because: (1) it is a quantitative indication of absorption, the range of which is correlated with the presence or absence of symptoms, as the case may be; (2) it is specific for lead when determined by the photometric dithizone method as described herein; and (3) when the "normal" range is exceeded, symptoms of intoxication are invariably present or will develop in the very near future. These three characteristics will not be found in any other clinical or laboratory finding, with the exception of the lead concentration in blood.

The lead level in blood has been found to fluctuate considerably during 24 hours. It may be high at night due to an increase in pH, or low during the day. Blood lead is the most accurate measure of absorption, but fluctuations may require

that two or more specimens be drawn, which results in considerable inconvenience.

Kaplan and McDonald[48] found the lead in blood to be 0.1 to 0.6 mgm. per 100 grams, in 24 of 26 children; and in 29 adult cases a result somewhat lower, all showing clinical manifestations of plumbism.

The lead content of feces is of little diagnostic value because there is no way to differentiate between lead which has been absorbed and excreted through the bile into the intestine, and that which has never been absorbed. A considerable amount of evidence has been offered which shows that most of the lead which is swallowed is excreted in the feces without ever having been absorbed. Therefore, the chief value of a high fecal lead is to show that a large amount of lead has been ingested within the past 24 hours or so.

The urine from a single micturition is not a representative sample but a 24-hour specimen will catch all fluctuations during this period. The volume may vary from 500 to 2,000 cc., slightly more or less. Lead may be reported as the total amount present in a 24-hour specimen, but a convenient standard is the concentration per liter.

It appears that the following rules should be established in diagnosing a case of chronic lead poisoning:

1. A detailed inquiry as to the place and length of employment, exact duties, and length of time off, if any.

2. A thorough physical examination by the physician for signs and symptoms of intoxication. Objective signs and symptoms are those which can be seen; others about which the patient tells are subjective. Both should be considered. The symptoms usually present are colic, constipation, vomiting, loss of appetite, loss of muscular strength, and pallor. A lead line on the gums may or may not be seen. Any other signs or symptoms should be recorded.

3. A "basophilic aggregation test" should be made for the total reticulocyte count. The normal is usually less than 1%.

4. A "basophilic stippled cell" count should be made, using Wright's stain. The normal is usually negative.

5. A lead determination should be made on a 24-hour specimen of urine. The "normal" ranges from about 0.01 to 0.07 mgm. per liter. The average is 0.03. A value of 0.08 to about 0.14 indicates either a mild exposure, or the recovery stage during several weeks' removal from an abnormal exposure. From 0.15 to 0.28 is indicative of intoxication. A value of 0.28 mgm. or more is a sure sign of a severe exposure and is a reliable indication of intoxication. In cases of chronic lead poisoning it has been found that lead may range from 0.08 to 0.76 mgm. per liter (and perhaps higher), the average being 0.28 mgm.

When the history of an abnormal exposure is established, the usual signs and symptoms are present, and lead in the urine is abnormal, then a diagnosis of lead poisoning is established. The basophilic aggregation test and stippled cells are used only to show blood destruction and regeneration, the cause of which must be confirmed by finding an abnormal amount of lead in the urine or blood. If either count is high it is of assistance in the diagnosis, otherwise not.

It goes without saying that a diagnosis is of little value unless the examiners are duly qualified, and the necessary precautions are observed. The examining physician should be thoroughly familiar with the various signs and symptoms of lead poisoning, and does not qualify as an expert unless he is experienced along this line. He should be well versed in the toxicology of lead, and either he or the technician must have special training in hematology. Blood smears must be kept free from dust and debris and should be examined within 24 hours. If kept too long the reticulocytes and stippled cells will break up and the count will decrease. The chemist must be duly qualified, preferably as a toxicologist. He must have the required training, and be thoroughly familiar with the detection and quantitative estimation of poisons in biological materials. If this experience is linked with a special study of the disease in human beings, he is qualified to express his opinion as to the toxicological significance of the quantities of lead found. Urine specimens should be collected in 2,000 cc. glass-stoppered Pyrex bottles, which have been rinsed out previously with hot 50% nitric acid, and then redistilled water. It is absolutely necessary that the employee's hands be washed before micturition, that his clothing be shielded properly, or removed, and that the bottle be kept away from the dusty atmosphere of the plant in order to avoid contamination with lead.

The Treatment of Lead Poisoning

PREVENTIVE. It is a foregone conclusion that the best way to avoid lead poisoning is to prevent it. The job of prevention reverts to the engineer, especially the ventilating engineer. It involves a considerable expenditure of money and close cooperation of employers, plant physicians, foremen, employees, and chemists. The installation of the following mechanical features and rules are of proven value:

1. Hoods with adequate exhaust ventilation at any point where dusts or fumes arise.

2. All mixing and shaking to be done in enclosed machines.

3. Transfer all powdered compounds by means of adequate vacuum lines.

4. Provide work tables with grated tops, ventilated from below. Equip with small narrow troughs filled with water, and attach to each side to prevent dust falling to the floor.

5. All inlets to exhaust ventilating ducts to be below the level of the nose, as far as possible, to prevent fine dust particles from being inhaled.

6. A chemical anaylsis of samples of air from various parts of the plant at different intervals during working hours. Any spot showing more

than 1.5 mgm. of lead per 10 cu. m. is a potential health hazard.

7. Employees in hazardous spots as found under (6) must wear respirators. Where movements are confined to a small area, forced draft respirators (fresh air from the outside) should be used. If the exposure is to fumes, masks must be provided with canisters charged with activated carbon. Respirators are only 70 to 90% efficient and must be inspected and changed frequently.

8. Allow no dust to accumulate on the floor.

9. Wet the floors at frequent intervals, and also powdered compounds if possible.

10. All cleaning to be done with large vacuum lines. No sweeping.

11. To prevent the collection of dust on rafters, sills, etc., install ventilating ducts equipped with dust filters near the ceiling or in mid-air.

12. To avoid the contamination of outside air and surrounding territory, and as an economy measure, install electrostatic precipitators in the stacks and flues.

13. The personal hygiene of the employees should be closely guarded. Provide locker-rooms with showers, a lunch-room isolated from the plant, and a change of overalls twice a day. Hands and face must be washed before eating, and there must be no eating, chewing or smoking during working hours.

B. Curative. It becomes the duty of the physician with the aid of the toxicologist to attempt a cure of those who have or will become victims of the hazard. Many therapeutic measures have been tried in attempting to cure the disease. The rationale behind any therapy involves either the demobilization, de-leading, or detoxification of lead in the individual. A brief discussion of each follows:

1. *Demobilization*—the conversion of lead into its most insoluble salt, the tertiary phosphate ($Pb_3(PO_4)_2$), and preventing its mobilization by depositing it in the bone as such. Aub and associates[19] were the first to advance the theory that demobilization could be effected by means of a high calcium diet and various calcium salts. The diet used included foods high in calcium, as milk, eggs, and vegetables. The calcium salt generally administered either orally or intravenously was calcium gluconate, but calcium lactate is frequently used. They obtained reliable evidence on experimental animals and human beings, that symptoms of poisoning were relieved and most of the lead was deposited in the bone. That the metabolism of lead ran parallel to that of calcium was established by them. In view of this fact, calcium gluconate and a high calcium diet has become almost universally used for demobilization and to relieve lead colic.

Aub, Robb, and Rossmeisl[24] later found that most of the lead in bone, particularly in relation to calcium, was found in the trabeculae and not in the cortex. Since it is known that the trabeculae act as a storehouse for available calcium, any lead stored here was also assumed to be available or easily mobilized. This was shown to be true by the disappearance of trabeculae and decrease of lead in leaded animals kept on a low calcium diet. As calcium is needed it is drawn from the trabeculae, and if lead is present a certain part of it is mobilized also. In the present work, 25 per cent more lead was found in the cortex than in the trabeculae of the sternum in Case 45, Table XII. This individual was known not to be suffering from lead poisoning. Most of the lead had been excreted or pushed away from the blood and deposited in the cortex as the very insoluble tertiary phosphate. In Case 25, Table X, an active case of lead poisoning, the reverse was found; 78% more lead was present in the trabeculae than in the cortex, indicating that most of it was available to mobilization from the trabeculae.

With due credit to the calcium treatment, the role of phosphate seems more important. We have no proof that calcium and lead form a complex molecule or radicle with each other in vivo. Instead of this we expect to find each forming its own separate salts, with some free calcium and free ionized lead. Then how can the administration of excess calcium bring about the precipitation of lead phosphate unless sufficient phosphate is present to take care of both? For this reason phosphate is as necessary as calcium, whether in the diet or administered in solution. Further evidence that the phosphate radicle has not received due consideration is found in the fact that the ratio of phosphorus pentoxide to calcium oxide in the ash of milk is 24:20, and in the ash of eggs 38.11.[62]

Calcium will relax smooth muscle and in this way the cramps accompanying lead colic are relieved, but this relief does not constitute a cure. The deposition of the phosphates of calcium and lead in bone is increased by the administration of calcium, but in the absence of sufficient phosphate it is obvious that this result would be impossible.

2. *De-leading*—converting lead in the body to a more soluble form, mobilizing it into the blood stream, thereby accelerating its elimination from the body.

Aub[19] found that this could be accomplished by a low calcium diet, decalcification, by producing an acidosis, or by alkalinizing the body.

A low calcium diet causes a calcium deficiency. The body draws on the storehouse of calcium in the trabeculae, and some of the lead deposited there goes along with it, apparently in a more soluble form, and is excreted. This has been demonstrated successfully, but a low calcium diet may be low in phosphate. When the trabeculae respond to the calcium deficiency in the blood, deposited phosphate also disappears. Therefore, a low calcium diet removes phosphate from trabeculae as well as calcium and lead.

Decalcification is brought about also by administering certain salts, potassium iodide being widely used. The effect depends upon the fact that the body tends to maintain a normal balance of its alkali reserves.

When potassium iodide is administered, the ratio between potassium and calcium is disturbed.

The body calls upon its calcium reserve to restore the balance in the fluids, with the assumption that lead too is again placed in circulation to be excreted. Some of the efficiency of this deleading process must also depend upon the fact that a part of the lead is converted into the more soluble iodide, and a possible stimulating effect on the thyroid. In maintaining a normal equilibrium, the acid reserve too or balance of the anions must be established. An excess of iodide might draw upon the phosphate ions and lead would be reabsorbed.

Acidosis is produced by giving certain salts which break up in the body to form acids which dissolve stored lead and hasten its excretion. Ammonium chloride is widely used. In the body it breaks up to form urea and hydrochloric acid. Other salts as the acid phosphates have been used but ammonium chloride is the most efficient. The mineral acids, such as hydrochloric and phosphoric, have been tried with less success.

Alkalosis is produced by the administration of an alkali such as sodium bicarbonate, which is very efficient. The effect is brought about in three ways: (1) excess sodium ions disturb the the calcium balance and there is a calcium and lead loss in the bone; (2) the normal pH (7.3) of the body may be slightly raised, causing some of the phosphates of calcium and lead to be dissolved; (3) the bicarbonate ions are in excess and phosphate ions may be drawn from the bone.

The most efficient de-leading salts are ammonium chloride, sodium bicarbonate, and potassium iodide, while the patient is kept on a low calcium diet.

3. *Detoxification*—the conversion of more soluble, more highly ionized lead compounds into the less soluble and less highly ionized, thereby reducing their toxicity.

For this purpose sodium thiosulfate has been used frequently. The rationale of this therapy seems to be based upon the idea that if it works in cases of arsenic and mercury posoning it will also be beneficial in plumbism. Solutions of thiosulfate containing traces of oxygen will become milky due to the formation of colloidal sulfur. Some hydrogen sulfide may be formed which will result in the precipitation of insoluble lead sulfide. From a chemical point of view this theory is unsound. The reaction is predominantly in favor of the formation of colloidal sulfur. The chances are that favorable results observed with this type of therapy are due more to removal from the exposure than to the treatment.

Magnesium sulfate and sodium sulfate cathartics are used chiefly for the relief of constipation by the dehydrating effect on the bowel, or to the fact that they hold water already present. The sulfate ion will convert lead in the bowel to insoluble lead sulfate and hinder its absorption. Sulfates are absorbed very slowly through the gut, so the conversion of lead to the sulfate in the tissues and fluids cannot be remarkable. Mineral oil and enemas are also used for flushing the bowel.

Shelling[57] studied the effect of dietary calcium and phosphorus on the toxicity of lead in the rat. He found that large amounts of calcium without phosphorus divert the available phosphorus in the body to form calcium phosphate, and thereby lessen the chance of the formation of lead phosphate. He believed that an abundance of phosphorus was necessary in order to deposit lead in the bone, or excrete it in a "deleading" process. A low calcium-high phosphorus diet was used to "delead." Leaded animals were found to improve much more on Na_2HPO_4 than on $CaCO_3$.

Gray[58] later used Shelling's findings in a study of leaded patients. Those kept on a low calcium diet, and phosphoric acid or sodium phosphate, were shown to excrete more lead. According to him they developed no symptoms during the treatment. They were apparently latent cases in which considerable time had elapsed since the last exposure.

Sobel, Gawron, and Kramer[59] found that vitamin D increased the concentration of lead in the blood and bones of rats fed on a diet in which lead was substituted for calcium. Animals receiving no vitamin D showed much lower concentrations. Sobel, Wexler, Petrovsky, and Kramer[60] showed that the concentration of lead in blood and bones of leaded rats was increased by vitamin D and a low calcium low phosphorus diet, and a high calcium low phosphorus diet, but a high phosphorus low calcium diet increased lead in the bone but lowered it in the blood. It is difficult to understand their last finding since lead is transported to the bone by blood, especially if the animals were sacrificed soon after the last feeding. They explain the beneficial effects of a high phosphorus diet "in terms of a lowered blood lead concentration," but this conclusion is not consistent with results obtained in the present paper. A child suffering from lead poisoning was treated with a high phosphorus normal calcium diet. The blood lead remained high but symptoms disappeared indicating detoxification and a persistent mobilization.

Di-basic sodium phosphate ($Na_2HPO_4.12H_2O$) appears to have the greatest therapeutic value in the treatment of lead poisoning of any compound proposed at this time:

(a) It will precipitate lead in its most insoluble form, the tertiary phosphate ($Pb_3(PO_4)_2$), and apparently this occurs to a large extent in vivo.

(b) Absorbed lead, and that which is unabsorbed in the gastro-intestinal tract, is therefore largely detoxified.

(c) The slight increase in pH (alkalosis) may mobilize some lead previously deposited in the bones.

(d) Excess sodium ions disturb the calcium balance, calcium and lead being drawn from the trabeculae.

(e) Mobilization and detoxification occur simultaneously.

(f) Free ionized lead is diminished by a depression of ionization by the phosphate which is known to be an excellent buffer.

(g) The salt is also a good cathartic, thus eliminating the use of others.

Until recently practically all patients admitted to the Cook County Hospital were immediately put on a high calcium diet with viosterol and intravenous calcium gluconate, if they were cases of suspected lead poisoning. Calcium gluconate or lactate (X to XXX gr. TID) was also given orally. In most patients the abdominal cramps disappeared, but the appetite, muscular strength, complexion, and steadiness returned to normal more slowly. The urinary excretion of lead before treatment was found to be abnormally high in all. In many cases this value was found to decrease somewhat, but never to normal, after one or two weeks on the high calcium, while in others the decrease was insignificant after two to three weeks. It was quite evident that the demobilization of lead and recovery was too slow to be remarkable. In view of this fact a decision was reached to try dibasic sodium phosphate together with a normal calcium diet. The results on three cases follow:

CASE F. G. A colored male was admitted to the hospital 12-13-37, complaining of severe abdominal cramps, general weakness, and constipation. He had a pasty complexion, lead line on the margin of the gums, and many basophilic stippled cells were seen in the smear with Wright's stain. He was employed by a lead smelting works for four years, shoveling and trucking scrap lead. There was a history of a previous attack 10 months prior to this. After four days on 30 gr. calcium lactate TID, a 24-hour specimen of urine was submitted in a brown glass bottle, the cleanliness of which could not be vouched for. The urine yielded .756 mgm. of lead per 1,000 cc. Subsequent treatment and results follow:

	Pb/1,000 cc. Urine
8 days on 30 gr. Ca lact. TID orally....	0.297 mgm.
1 day on 60 gr. Na_2HPO_4 TID orally....	0.183 mgm.
3 days on 60 gr. Na_2HPO_4 TID orally..	0.132 mgm.
7 days on 60 gr. Na_2HPO_4 TID orally..	0.113 mgm.
3 days on 15 gr. Ca lact. TID orally....	0.139 mgm.

Observe that the initial calcium treatment did not demobilize lead remarkably, the urinary concentration remaining above the average of 0.28 mgm. found in 40 other victims. At this time the patient stated that his cramps were less severe but otherwise he felt about the same. After the phosphate treatment was started, the concentration steadily dropped to a point where it was still slightly abnormal, and then showed a slight increase when calcium again was administered. After the third day on the phosphate, blood calcium was 10.45 and phosphorus 3.95 mgm. %. The patient stated that he felt much better on the phosphate treatment, and even asked for it. The attending physicians concurred in the opinion that recovery was practically complete. He was discharged three weeks after admittance, and told not to return to the lead work.

The writer was not of the opinion that he was "cured," because he still seemed to be slightly weak and unsteady, but had no pains. Given the proper diet, rest, and fresh air, it is evident that recovery would be better within a short time.

The results in this case indicate that lead was detoxified as it was mobilized from the bone. The decrease in the urinary excretory rate could indicate demobilization, but it is more likely that mobilization of available lead from the trabeculae was practically complete at this time.

CASE J. C. A white male entered the hospital 3-28-38 complaining of colic, constipation, vomiting, loss of appetite, general weakness, and nervousness. He was extremely jittery, had a marked pallor, a very distinct lead line on upper and lower gums, and two or three stippled cells per field were seen. He had handled pig lead and transferred white lead to barrels for eight months. A brief of the treatment and results follows:

	Pb/1,000 cc. Urine
3 days on 10 gr. Ca lact. TID orally....	0.306 mgm.
2 days on 7½ gr. Na_2HPO_4 intravenously, once a day.....	0.256 mgm.
2 days on 15 gr. Na_2HPO_4 intravenously	0.241 mgm.

Here again the calcium relieved the cramps but the patient and physicians agreed that the greatest benefit was derived from the phosphate. There was some hesitation about injecting the phosphate intravenously, due to the possibility of raising the pH too high, creating a negative calcium balance, and laking the blood. However, 10 cc., and, later 20 cc. of a 5% sterile solution was injected very slowly with no noticeable untoward effect. In the meantime the patient was kept on a normal calcium diet. The urinary excretion of lead does not show a marked decrease, indicating that demobilization in the bone was very slight if at all. The fact that the excreted lead was approximately in the same range as before treatment was started, indicated mobilization, and that the circulating lead was largely detoxified because the symptoms had subsided. He regained his appetite, color, and strength and was released two weeks after admittance.

CASE B. A. A two year old colored boy was admitted to the hospital February 18, 1938, with encephalophathic changes. The mother had burned storage battery cases as fuel at different intervals. The child had a full set of teeth. There was a lead line on the upper and lower gums, and a bluish-black deposit on the inner surface of many teeth. The x-ray revealed lead lines in the epiphyses of the long bones. At different intervals he was fretful, irritable, and nervous. He was subject to convulsive seizures and at times became drowsy, refusing to eat or play with toys. There was a pill-rolling motion with the fingers. The consensus of opinion from the history of exposure and clinical evidence was that the patient was a victim of lead encephalopathy as indicated by a brief of the treatment and results:

	Pb/100 cc. blood
No treatment	0.188 mgm.
2 wks. on 5 gr. Na_2HPO_4 TID orally	0.124 mgm.
5 days on 15 gr. Na_2HPO_4 TID orally	0.116 mgm.
*10 days on 15 gr. Na_2HPO_4 TID orally	0.134 mgm.
2 days on 2 gr. Na_2HPO_4 once a day, intravenously	0.122 mgm.
2½ days on 3 gr. Na_2HPO_4 twice a day, intravenously	0.149 mgm.
35 days on 15 gr. Na_2HPO_4 TID orally	0.227 mgm.

* Blood calcium here was 15.5 mgm. per cent, phosphorus, 5.5, said to be normal for a child of this age.

The child was remarkably improved 11 weeks after admittance. In the opinion of the pediatricians it could have been released (with proper instructions to the mother) before this, but was held longer to see if the lead level in the blood could be decreased to normal. According to them, most of the encephalopathic changes had cleared up. This was quite evident but there still seemed to be a slight amount of sluggishness present and an abnormally slow blinking of the eye lids. Viosterol was administered with a normal calcium diet at the same time. Ten cc., and, later, 20 cc. of a 1% sterile solution of the phosphate was injected intravenously very slowly with no untoward effect.

The disappearance of symptoms during the phosphate treatment while the concentration of lead persistently remained at an abnormally high level seems to be sufficient proof that the circulating lead was detoxified. It also indicates that lead was mobilized, instead of demobilized, and that there must have been an enormous storage of lead during the exposure, thus necessitating a longer time to liberate and excrete it.

As a supplement to this work, in the future specimens of blood will be obtained in definite cases of plumbism, before and after the phosphate therapy. By dialysis, an attempt will be made to see if the ionized lead in the dialysate is significantly decreased by the phosphate. Polarigraphic readings on whole blood, blood serum and dialysate may give some indication as to what form lead exists chiefly in the circulating blood.

We must remember that no matter what salt is used, nothing works the same in vivo as in vitro. Nothing is absolutely insoluble. It is expecting too much to believe that excess phosphate or calcium will convert lead quantitatively into the tertiary phosphate or any other phosphate in vivo. It is possible and sufficient evidence has been produced to show that the reaction is in favor of this. However, a small amount of that lead unavoidably remains in solution as the chloride, lactate, or some other salt.

Taeger[56] believes that massive doses of any salt will liberate stored lead, and that even calcium should be given in very small amounts at first and increased gradually over a long period of time. It should be administered in accordance with the degree of hypocalcemia or hypercalcemia. Even the diet should be carefully watched and only gradually returned to normal. Perhaps this is the ideal treatment, but it is doubtful if it holds any advantage over high phosphorus normal calcium.

Industrial physicians often ask the question, "Is it advisable to try to de-lead a man while on the job or should the lead be demobilized?" From the results obtained with di-sodium phosphate, the answer is that de-leading on the job would appear to be entirely safe, if this drug is used. Provided that the proper mechanical methods of prevention have been installed, that the man is maintained on a normal calcium diet with the necessary vitamins, and that there is no hypocalcemia, it is evident that di-sodium phosphate will prove to be very beneficial. It may be administered in aqueous solution in 10- to 30-grain doses three times a day. The effects will be the relief of constipation by catharsis; a diminution of lead absorption from the gastro-intestinal tract by formation of lead phosphate; the storage of lead in bone is largely prevented by a change in the alkali reserve, which is equivalent to a mobilization of lead; di-sodium phosphate detoxifies lead in the circulation, probably by the formation of colloidal lead phosphate; excretion keeps pace with absorption and accumulation of lead ceases. It is realized that a study of a larger group of patients would be more conclusive, but three human cases are more conclusive than many more animal experiments.

Suspected cases of poisoning have been sent to farms to recover, and the rotation of jobs is very beneficial. There is no doubt that removal from the exposure, fresh air, and an adequate diet is very necessary.

If we could be sure that a patient would not develop some condition within two or three years which would liberate toxic amounts of stored lead from the bone, it would be safe to let nature take care of the gradual elimination of lead. Since this is not possible, it is safer to liberate stored lead in detoxified form by the use of di-sodium phosphate than to take the chance of nature mobilizing it by acidosis in a more toxic form.

Summary

THE "normal" concentration of lead (mgm. Pb per 100 grams) has been determined in the tissues of 25 individuals, and in the urine of 18. The average values are as follows:

5 fetuses:	liver 0.036, kidney 0.021, bone 0.052 mgm.;
5 children:	liver 0.18, kidney 0.09, bone 1.54 mgm.;
5 youths:	liver 0.15, kidney 0.08, bone 1.07 mgm.;
5 middle-aged:	liver 0.23, kidney 0.10, bone 1.07 mgm.;
5 old-aged:	liver 0.10, kidney 0.06, bone 1.35 mgm.;
18 adults:	Urine 0.03 mgm. Pb per 1,000 cc.

Other workers have determined the "normal" lead in blood as 0.03 mgm. per 100 cc.

The above values are considered "normal" because the subjects had no excessive exposures to lead and no indications of lead intoxication. The source of this "normal" lead is food, drink, and the atmosphere, which is unavoidably contaminated with small non-toxic quantities of lead under ordinary living and working conditions. Human beings are born with traces of lead derived from the body fluids of the mother. As soon as they begin to breathe, eat, drink, work, and play, the ab-

sorption of lead increases until values of approximately 0.2 mgm. in the liver, 0.1 in the kidney, and 1.3 in bone are reached. These levels appear to be maintained throughout life, indicating that excretion is keeping pace with absorption, the accumulation of lead ceases, and normal equilibrium is maintained.

The photometric dithizone method has been made specific for lead, and is the most reliable method for the purpose.

When the exposure to lead becomes excessive or abnormal, absorption exceeds excretion, and the equilibruim is disturbed. Lead accumulates in the tissues to the extent that there will be a cumulative effect when a certain range is reached.

Fifty-one fatal cases of suspected lead poisoning were studied. Nine cases of non-industrial chronic lead poisoning in children yielded the following average concentrations: 3.13 mgm. in the liver, 1.43 in the kidney, and 5.38 in bone. The exposures were due to pica, and ashes from old storage battery cases used as fuel. These findings were correlated with definite symptoms of encephelopathy due to lead. (Case R.D. was added later).

In 21 industrial cases a justified conclusion of chronic lead poisoning was reached because the average lead concentrations of 0.97, 0.58, and 5.47 mgm. Pb. per 100 grams of liver, kidney, and bone respectively were correlated with abnormal exposures and well-defined symptoms of intoxication. The total lead content of a 300-gram composite of 100 grams each of liver, kidney, and bone was found to be about five times the "normal."

A conclusion of lead poisoning was considered doubtful in five cases (except the colloidal lead phosphate case) because the exposures and symptoms were vague or doubtful. The average concentrations were 0.31 mgm. in the liver, 0.36 in the kidney, and 1.68 in bone. The total lead content of a 300-gram composite specimen (mentioned above) was about twice the "normal."

In 13 cases a conclusion of lead poisoning could not be justified because the exposures were very slight or almost negligible, and the symptoms were closely associated with some demonstrable pathology, or external injury. The histories ran parallel to the following low average lead concentrations (which were approximately the same as the "normal"): liver 0.19 mgm., kidney 0.11, and bone 0.93. (Case J.B. was added later).

In one acute case the chemical findings left no doubt that the woman was acutely poisoned with lead, but the history was of such a nature that it could not be said that lead was the immediate cause of death.

Forty-six cases of non-fatal lead poisoning are reported.

Five children showed definite encephalopathic changes, and concentrations in the blood ranging from 0.06 to 0.32 mgm. per 100 cc.

Forty-one men employed in the lead industry revealed signs and symptoms indicating lead intoxication, and these were correlated with lead concentrations in the urine ranging from 0.08 to 0.76 mgm. per 1000 cc., the average being 0.28.

It seems that entirely too much confidence has been placed in the "basophilic aggregation" test and "stippled" cell count in the diagnosis of the disease. Since there are no signs or symptoms pathognomonic of lead alone, the determination of lead in the urine or blood of living victims and in the tissues of the dead appears to be the most reliable diagnostic feature. The clinician may suspect the disease from his evidence, but the diagnosis is incomplete without the chemical findings.

Di-sodium phosphate is proposed as the best therapeutic agent in the treatment of the disease. The effect appears to be a detoxification of lead as the patient is "de-leaded." The drug should be used in conjunction with a normal calcium diet to avoid a hypocalcemia, and is contra-indicated in diarrhea and achlorhydria.

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Chile Activates Prevention

ENLARGING the building to take care of the ashes seems to be the public's slow method of meeting many situations. Voting funds for prevention comes only after repeated exhortation and demonstration. How different it is in private endeavors where most businesses would collapse otherwise. The principle of insurance to meet the cost of disasters is accepted, but planning expenditures to prevent them goes begging. Still, in the public health and welfare fields some progress has been made, and with remarkable results, as witness smallpox, typhoid fever, diphtheria and even tuberculosis.

In regard to tuberculosis the Detroit Plan,* adopted late in 1936, encompasses the idea of appropriating an amount equal to about one-fifth of the annual hospital bill (of about $1,000,000) for a carefully schemed program of prevention. Already the Health Department was spending $180,000, or 12% per capita, for the prevention of tuberculosis, and the Division of Health Education $30,000 more. It is not our purpose to discuss here Detroit's tri-partite plan of attacking this problem through cooperation of medical, health, education, and political agencies by which some 400 cases representing otherwise hidden foci of infection were corralled in 1937, in addition to the average yearly number of about 2,000 new cases reported. The Detroit plan and results can be found elsewhere.*

Instead, we wish to call attention to what is proceeding over the Equator in one of our South American countries — Chile.** Despite a compulsory insurance scheme of some 15 years' standing against sickness, old age, invalidity and death, neither sickness nor death had fallen off to a degree justifying the expenditure incurred. Recent investigations showed that reported sickness was far below actual sickness found by medical examinations of supposedly well persons. The result has been the usual disproportionately high death rate. In one study of 16,078 manual workers it was found that over one-fifth required immediate treatment for syphilis (14.8%), cardiovascular disease (4%), and tuberculosis (3%). In another study of 1,400 students supposedly well, 182 were found diseased—cardiovascular disease 58, tuberculosis 41, and syphilis nine. In general, these three diseases were found to comprise two-thirds of the real morbidity in Chile.

Chile has some 1,185,000 insured persons on the average, but 285,000 of these are constantly on gratuitous benefit for disabilities, which means a tremendous burden. Furthermore there is the question of those dependent upon the disabled insured. A study in Santiago showed that only 23% of 2,119 persons in all stages of tuberculosis were finally able to return to work while 76% of 569 persons in early stages, who were treated in the same city, were rehabilitated. The average case of syphilis was found curable for 250 pesos if taken in time but to cost 10 to 20 times this much if treated too late.

A further study showed the following possibilities in prevention as regards both disease rates and death rates: (1) Infectious and some occupational diseases are responsible for about 20% of illness; (2) metabolism diseases, very largely preventable, for 20 to 25%; and (3) tuberculosis of the lungs, syphilis, and cardiovascular diseases for another 50%.

After eight months of Parliamentary discussion, a law was enacted to implement effective economic and social aid for diseased persons, both those capable of being restored to work and those who would never work again. It was concluded that it is not the object of social legislation simply to protect the sick and old, by which man is considered as working for the single purpose of collecting enough money to permit him to find rest, as such contradicts the physiological and psychological characteristics of man. Instead it was concluded that work should not exceed the individual's capacity for it, must remain related to his will, and must have some attractive feature. The solution does not lie in the progressive abolition or gradual shortening of wage labor, but in reform-

* VAUGHAN, HENRY F., and DOUGLAS, BRUCE H.: Intensive Case Study Work in Tuberculosis, *J.A.M.A.*, 109,10:771-777 (Sept. 4) 1937. Also, *City Health*, The Bulletin of the Detroit Health Department, XXII, 1:3-10 (January-July) 1938—"Public health in Detroit, annual report 1937."

** EDUARDO CRUZ COKE L. (Minister of Health, Public Assistance, and Social Welfare): The Chilean Preventive Medicine Act. *International Labour Review*, XXXVII, 2:161-189 (August) 1938.

ing it and adapting it to the functional capacity of the individual. A state of disease in a worker starts a vicious circle and, for him, two courses of action have been open—to continue to work the full day, or not to work at all. In either case he and his family suffered and ultimately became a charge on the State. It was felt that the answer was to fortify the worker economically so that he could make the most of his abilities, whatever they were, and at the same time not be an evil influence on his colleagues and on production. It would be much to the employer's interest to eliminate employees whose output does not reach a certain level, even if he has to continue payment of wages in full. Partial compensation for sickness, invalidity, old age, and death is not the solution. Financial provision should be subordinated to social provision and should follow and not precede it. In short, disease should be prevented.

The Chilean solution, beginning with the year 1938, provides: (1) Systematic periodic compulsory medical examination for the members of all social insurance funds; (2) total or partial "preventive rest" with full wages for a period determined by the possibilities of re-entering into employment; and (3) the financing of these two schemes—the former by means of a levy of 2½% on the gross income of the funds, and the latter by a contribution equal to 1% of wages paid by the employer. Both the levy and the contribution may be increased if need be by 50%, by Presidential decree.

The principal measures of enforcement are: (a) The insured person loses all his right if he refuses to be examined; (b) the examinations are conducted in accordance with certain specified standards; (c) any treatment imposed is compulsory, especially for syphilis, and refusal to submit is punishable as under "a"; (d) professional discretion must be observed strictly and a doctor who violates this rule may be punished severely—this to prevent employers from choosing the most healthy workers; (e) the funds may combine for medical purposes of any sort; and (f) the funds are authorized to acquire the sanatoria and convalescent homes necessary for the working of the Act—the interest on the sums spent in this way to be paid out of the levy of 2½% on gross income.

Regarding the medical policy under the Act, it is recognized that efficiency depends upon the reliability of the medical examination and the validity of the standards adopted for same. Hence "planned medicine" was initiated, principally to do away with expensive and capricious tendencies. "Neither the medical examination nor the treatment resulting from it should depend upon the individual judgment of each doctor," but upon an investigation restricted to the discovery of what steps are most effective. By such procedure there has already resulted a saving of over 1½ million pesos a year through the prohibition of useless medical treatment.

The fund has a million members and a total annual receipt of 150 million pesos from which the levy of 2½% collects four million pesos for medical examinations. While this sum appears small, it is calculated to be sufficient since equipment is already at hand and the discovery of hidden disease will effect tremendous saving of the waste previously incurred in attempted cures and premature deaths. The funds created also provide for a cheap housing program, which, from rentals and sales, is accumulatively amortized.

Despite the great distances separating persons in agricultural areas, it is estimated that some 600,000 workers will be examined during the current year. In the towns they are being examined at their workplaces and in the country whenever they report to the local doctor for some slight illness. Also several rural dispensaries have been set up. Experience already has shown that 40% of persons seeking treatment are not genuinely sick or that their afflictions are of no importance.

It is felt that any system of occasional benefits is so unfit as to be useless in those cases in which it is most required.

For "preventive rest," provision is made for an average of six months per case, and inquiries already made have shown that about 2% of the workers in Chile require rest more or less urgently. To meet the situation in the first year, funds become available for 800 to 1000 beds in sanatoria.

Chile has about 60,000 salaried employees for whom 2½% of the levy will allow about four million pesos per year, enabling medical examinations to be made twice a year at 20 pesos each, full sanatorium expenses to 200 patients, interest on the fund invested, treatment of specific diseases like syphilis, home visits, and other unforeseen contingencies.

"The central point of the campaign against avoidable morbidity may thus be found in the medical examination, preventive rest, and curative action, introduced by the new Act." Coordinated with this is the necessary social investigation in regard to tuberculosis and syphilis.

Chile has had to take its actuarial calculations from British, Danish, and German experience which, though quite dissimilar to its own, are believed effective for the reduction of hidden disease rates.

We shall await with interest the results of this determined procedure in our neighboring American republic. It involves a country where over half of the deaths occur before 15 years of age, and over 53% of those over that age die of tuberculosis, cardiovascular disease, and syphilis. Superficially the case appears different in the United States. With us, the general death rate is comparatively low and continues to improve annually, while that for tuberculosis has dropped about two-thirds since the turn of the century, but statisticians show unusually high rates from various causes among unskilled and semi-skilled workers and, indeed, for tuberculosis in early childhood and the age group 20 to 40. Economically and socially our present solution is too universally "hind side before," and we need to treble our efforts "to put out the fire instead of enlarging the building to take care of the ashes."

INDUSTRIAL HEALTH AND DISEASE

The Eye and Ear in Industrial and Social Life

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IN SELECTING this subject for presentation, I had hoped to get something different from the usual eye, ear, nose and throat paper of our southern section, and I feel that in a way I have succeeded; however nothing in my discourse is original, everything being taken from past records, statistics and papers.* In the preparation of this article I was amazed at the great amount of material available, and I have only attempted to cover briefly and in a more or less general way some of the more vital points as I see them.

The Eye

IN EVERY type of industry, except the blind trade schools, the vision is of vital importance. Practically all companies at the present time have all workers examined before their services are accepted and regular examinations given frequently to see that proper physical standards are maintained. If a prospective worker has poor vision he or she certainly has much more difficulty in securing a position. In every kind of factory or machine works, the individual who does not see normally becomes a serious menace to others in the same working area, and most likely to suffer injury of self, seriously or even fatally.

Stereoscopic vision is so valuable that persons with one eye must be excluded from many kinds of work, such as engravers, goldsmiths, airplane pilots, railroad employees, chauffeurs, and from all military activities, and many other forms of industrial service. Man is the only animal, except monkeys, who has what is called binocular single vision, that is, he can tell not only the direction of an object but fairly accurately its distance by the use of both eyes pointing at the same object at the same time like two range finders. Other animals do not use both eyes on the same object at the same time. The outside limits of vision of man are greater than half of the space in which he is placed and his eyes can move accurately, with the greatest precision to any object in any part of his field of vision, and such movements may be supplemented by movements of the head and body. He sees distinctly only that at which he looks directly. Peripheral vision assists him in discovering moving or other objects to which, if he wishes, he may direct his gaze. Poor vision in an eye may save a man from accident caused by some moving object, as a car approaching from the side. Therefore the individual with two eyes, even if the vision of one is poor, is more likely to escape injury than the person with only one eye. One-eyed people are always more likely to meet with accidents than those having two normal eyes.

Every oculist knows that many people consult him and though they have not seen out of one eye for many years or since birth they did not know this until examined. Often the patient to his astonishment discovers that in one or perhaps in both eyes he or she has only a small percentage of normal vision. If this person with one eye seeing only one-twentieth as much as the other eye should have a slight accident to the bad eye, he or she would be inclined to ascribe the loss of vision to the accident, no matter how trivial it might be. If he or she were a malingerer and had known that the sight was previously defective in one eye, he or she could attribute the loss of vision to the possibly harmless accident. To determine how much a given accident or disease has injured the eyesight, the vision of the eye or eyes before the accident and after must be known. Therefore when any industrial organization or state undertakes to pay for the results of an injury to an eye, they should have a record of the vision of each eye of every assured or employed person. This will insure fairness to both parties. Also the examination will discover many who have defective vision and who are more likely to receive injury, not only to eyes, but to other parts of the body, and possibly loss of limb or life.

Persons who have narrowed or constricted fields of vision would be unable to protect themselves from objects moving towards them outside of the space in which they can see; naturally such an individual should not be employed in certain dangerous vocations, not only for the protection of the individual, but for the employer, and also for the protection of fellow workers or others.

Those whose vocation requires good vision for small signals, colors, such as trainmen, motormen, airplane pilots, army and navy service, are subjected to special quantitative and color tests. It is known that the use to excess of certain drugs, or alcohol and tobacco, may destroy the color perception of small or distant objects without having much effect on general color perception.

The first aid service with trained nurses and doctors is vitally important to any industry to maintain proper physical standards.

In most industries so much depends upon the good vision of the employees that efficiency as well as the prevention of general accidents requires the protection and preservation of eyesight. In certain trades many eyes are injured or destroyed by flying particles, molten metals or corrosive compounds. Most of these accidents can be prevented by correctly fitted protective goggles. It is practically criminal negligence in not using protective glasses that causes the loss of many good eyes, robbing the worker of his usefulness and often throwing him upon his friends or the local government for financial assistance. Education of the workmen and their directors often takes much time and patience before full cooperation can be secured. In certain trades the illumination should not be reduced too much; ordinary glass reduces it nearly 7%, and if isinglass is placed behind the lens the illumination is reduced about 12%. Fortunately at the present time correctly made and tinted protective lenses are easily secured.

The Ear

IN CONSIDERING the ear in industrial workers, we think in terms of boiler-makers' deafness, airplane deafness, and cloth weavers' deafness. We know the sense of hearing, while perhaps not as vital as vision, is very necessary for any worker to be able to win out in the rapid mechanical productive work of this age, and it becomes the serious duty of every industry to prevent occupational deafness by the elimination as far as possible of all excessive or pathology producing noises.

Occupational noises are divisible into two classes: First, those of sudden access and magnitude, such as gun reports, explosions of shells, blasting; these cause deafness by immediate effect on the cells of Corti's canal in the cochlea, with or without middle-ear injury and rupture of drum. Second, there are sounds of prolonged duration, sounds of high pitch and full of overtones, e.g., those to which boiler-makers, coppersmiths and many machinists are subjected, and which produce their effect by stimulation of, and followed by degeneration in Corti's organ, first in the ciliated cells and progressing to the nerve fibers. Such cases of de-

* Read before the Fifty-Ninth Annual Meeting of the Louisiana State Medical Society on May 4, 1938, at New Orleans; published in *New Orleans M. & S. J.*, October, 1938.

fective hearing are often accompanied by tinnitus and in more advanced cases by vertigo, especially where vibration is present.

The pathologic proofs for these statements are based on experiments on animals. Guinea pigs were used and one or both ears were subjected to continuous notes or tones for varying periods up to 14 days, then the animals were killed and their cochlea examined. Wittmarck was one of the leaders in this pathologic research.

Ritchie Rodgers some years ago investigated boiler-makers' deafness and his conclusions were that loss of hearing for high notes is not the inevitable sequel of noise deafness as had often been thought, but rather that loss of hearing is most marked in that portion of the cochlea corresponding in pitch to the noise to which the individual has been exposed; in other words, the deafness, whether it be most marked for low, medium or high notes, corresponds in pitch to the noises among which the individual works. Later, however, the loss of hearing for high tones, if not previously present, becomes marked, owing to the greater proximity and vulnerability of the basal portion of the cochlear canal. Those supporters of the theory that the basal membrane acts as a resonator cite these experiments on animals and the clinical findings of Ritchie Rodgers as strong evidence in favor of the view that sound is analyzed in the cochlea, in so far as it is analyzed at all.

In occupational deafness of the first variety, sudden atmospheric condensation incident to explosions may bring about rupture of the drum. However, such injuries usually heal rapidly. It is interesting to note that when rupture of the drum does take place, the effects on the internal ear are less and more quickly overcome.

In airplane pilots acute sense of hearing, while an excellent asset, is not considered quite as important as perfect vision. In commercial aviation, the United States Department of Commerce physical requirements on a basis of normal 20/20 whispered voice test, require about one-third normal hearing in each ear.

Pilots are subject to three types of deafness: First, deafness due to rupture of ear drum, most often caused by rapid descent, directly due to unequal barometric pressure on either side of the tympanic membrane, and often associated with obstruction of eustachian tube; second, the deafness due to the continuous sharp reiterated crack of the exhaust; this is practically always of a temporary nature, and often lasts several hours after a flight. The third type is that affecting those who test airplane motors, this is usually temporary, as the workmen are only exposed to extreme noises for short intervals every day with frequent changes.

Occupational deafness occurring in civil life affects boiler-makers, black-

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smiths, locksmiths, mechanics, engineers, weavers and stokers, and is most often characterized by progressive diminution in the hearing perception accompanied by tinnitus due to excessive irritation of the cochlear branch of the eighth cranial nerve. Gottstein and Kayser found marked impairment of hearing in 50% of smiths and machinists. Holt found marked deafness in 35% of coppersmiths; Marr found that 91% of boilermakers had impaired hearing. Haferman, in a limited number examined, found no boiler-maker with normal hearing. McKelvie, in thousands of weavers examined, found 24% with some degree of defective hearing.

In industry defective hearing may be due, not to noise alone, but to other factors. Certain groups of workmen in chemical plants or other allied branches may become more or less deaf due to certain poisons; this is especially the case in chronic poisoning due to lead, mercury, arsenic, phosphorus, sulphur, coal gas products and aniline dye products.

The prevention of deafness in industry is at present being carried out by the owners of plants and the workmen aided by trained nurses, doctors and technical experts. Industrial safety and efficiency, which touches our whole social structure, is not only a matter of machines, structures, foolproof devices, signs and systems, but in the final analysis is dependent on the education and intelligent, unselfish and willing cooperation of the human factor.

The social problem of the care and prevention of blindness and deafness is one of great magnitude and has many ramifications, and I will very briefly mention some of the factors. A recent national health survey by the U. S. Public Health Service gives the following statistics. Blind in one eye, 34 per 10,000 persons; partial blindness, 20 per 10,000 persons; blind in both eyes, nine per 10,000 persons. Deafness: Partial deafness, 119 per 10,000 persons; total deafness, five per 10,000 persons. In the public schools of the U. S. there are about 30,000,000 children, and the records of nearly 1,000,000 examined in 1935 - 1936 showed over 5% had poor hearing. Of this number examined, over 22,000 were treated by physicians and 4% were restored to normal hearing, and over 4% were greatly improved. Considering these figures as approximately correct, there are in the U. S. 1,500,000 children with some degree of hearing defect, and, compared with past cases treated, of these 1,500,000 children, 60,000 could have their hearing restored to normal, and an equally large number could have their hearing improved with proper treatment.

Summary

THOSE unfortunate individuals whose sight and hearing are gone are automatically forced out of every day social life, and quite often when a person suddenly becomes blind or deaf, the shock is acute, and complete

mental breakdown may be the end result. The blind and hard of hearing societies in every city of any size, with their club-like meetings and their trade schools, are doing splendid work. Every state in the union has some form of schools or institutions for the blind and deaf, and every year millions of dollars are used in making these individuals happy and often self-supporting.

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Traumatic Appendicitis

IT IS generally conceded, at least by industrial surgeons and insurance companies, that appendicitis of traumatic origin is a disease entity*. Of course it is impossible to prove in the laboratory or by any other means that in a certain case acute appendicitis is traumatic in origin. However, in going over closely the history and clinical course of many cases reported in the literature as instances of traumatic appendicitis, one must admit that coincidence alone would be overtaxed to explain the phenomenon.

This case is not reported with the intention of either supporting or denying the existence of traumatic appendicitis. Most authorities will admit that trauma may be a contributing factor in the production of acute appendicitis in a previously diseased appendix. Since no organ in the body is immune to injury no matter how deeply seated, it is not logical to say that appendicitis on a traumatic basis canot occur in a previously normal appendix.

No attempt will be made in this report to review the literature on traumatic appendicitis. That has been

*C. M. BURGESS, M.D., Honolulu, Hawaii, in *J.A.M.A.*, August 20, 1938.

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so ably done recently by Maes and McFetridge[1] and by Shutkin and Wetzler[2] that further comment would be repetition. However, reading of the literature makes one thing apparent. For the cases reported as instances of traumatic appenticitis the mortality rate is extremely high. In Kelly's[3] series of 50 cases it was 50%; 37 of the appendixes had ruptured or were gangrenous. In 16 of the 25 fatal cases appendical involvment was unrecognized. In all subsequent series of cases the proportion is about the same.

Traumatic appendicitis is so rare that no one has a large series or becomes proficient in its care. But by physicians bearing the possibility of its existence in mind perhaps the mortality and morbidity of the disease process can be lessened.

The mechanism of traumatic appendicitis has not been satisfactorily explained. Gangrene and rupture predominate as a pathologic process, but no differentiation can be made from stained sections. The diagnosis must be made from the history and clinical course. The weight of opinion would indicate that the condition is due to a sudden change in intra-abdominal pressure which causes an influx of cecal contents past a narrowed portion of the appendical lumen. The distention and subsequent edema cause complete obstruction. Ultimate destruction results from rapid bacterial growth.

The patient, a man aged 46, a Hawaiian, was first seen about 10 a.m. May 21, 1937. Physical examination revealed that he was very well developed and well nourished and apparently in considerable pain. The right lower quadrant was extremely tender and rigid. The temperature was 101 F., the pulse rate 88 and the respiratory rate 23. The white blood cell count was 22,000, with 88% polymorphonuclears. A diagnosis of acute appendicitis was made, and at operation a ruptured appendix found. The history is of great interest. For the previous two weeks the patient had been using a pneumatic drill to raise the ceiling of a tunnel through stone. This drill was suspended from the ceiling by means of a strap. To exert pressure he rested the butt of the drill in his right iliac fossa. About 10 a.m. May 20, 1937, while drilling, he suddenly had a pain in the right lower quadrant so sever that it doubled him up and prostrated him on the floor. This lasted several minutes, and, although it then became less severe, it prevented further use of the drill in the former position. A dull pain persisted until the next morning, when at work it became so severe that he was sent to a physician. There was no previous history of pain in the right lower quadrant.

The observations at operation were unusual. The cecum was attached unusually far toward the midline. The appendix was ruptured in such a way that the tip was amputated. It lay to the right of the cecum, sharply kinked upward at the base and directly over the psoas muscle. It was tightly bound to the posterior part of the parietal peritoneum by old tough adhesions at the region of the sharp kink upward. The peritoneum was drained. Convalescence was uneventful. Compensation was allowed.

This case seems to fulfill all the requirements necessary for the diagnosis of traumatic appendicitis. It is presented because of its rarity and because physicians cannot keep too much in mind a condition so high in mortality and morbidity.

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